IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF OKLAHOMA

Case No. 4:05-CV-329-GKF(SAJ)

STATE OF OKLAHOMA
Plaintiff

v.

TYSON FOODS, INC., et al.,
Defendants

Herman J. Gibb, Ph.D., M.P.H.
Tetra Tech Sciences
Arlington, VA 22201

Herman J. Gibb — **Herman J. Gibb**

December 1, 2008 — **Date**

INTRODUCTION

1. I was asked to evaluate the opinions of consultants for the State of Oklahoma regarding allegations of health effects caused by poultry litter in the Illinois River Watershed of Oklahoma.

QUALIFICATIONS

2. I am President of Tetra Tech Sciences (formerly named Sciences International), a health risk assessment consulting company in Arlington, Virginia, and an Adjunct Professor in Environmental and Occupational Health at the George Washington University School of Public Health and Health Services in Washington, D.C.
3. I have a PhD in Epidemiology from the Johns Hopkins University and an MPH in Environmental Health from the University of Pittsburgh.
4. I currently am a member of the World Health Organization's (WHO) Foodborne Epidemiology Reference Group (FERG). The purpose of the Group is to develop an estimate of the global burden of disease from foodborne illness. I chair the Chemical Task Force of the FERG and am a member of the FERG's Source Attribution Task Force. The Source Attribution Task Force is assessing the contribution of bacteria, viruses, parasites and chemicals to the burden of disease by different routes of exposure including food.
5. Prior to joining Tetra Tech Sciences in January 2004, I had a 29-year career at the United States Environmental Protection Agency (EPA). Most of my career was spent at EPA's National Center for Environmental Assessment where I served as Associate Director for Health, Assistant Center Director, and staff epidemiologist.
6. I received EPA's Gold Medal for Exceptional Service for my risk assessment analysis of the epidemiologic studies on arsenic in drinking water, EPA's Scientific and Technological Award for senior authorship of an epidemiologic study on chromate production workers, and EPA's Award for September 11 Activities for membership on the World Trade Center Particulate Matter Toxicological Assessment Team. I directed EPA's health and exposure assessment of the ambient pollution which resulted from the collapse of the World Trade Center. I served on two White House Interagency Committees on health risk assessment and a White House Interagency Committee on mercury in the Gulf of Mexico.

7. I am a co-author of the WHO Environmental Health Criteria Document on the *Principles for the Assessment of Risks to Human Health from Exposure to Chemicals* (IPCS 1999) and of EPA's *Risk Assessment Principles and Practices* (EPA 2004a). I am the author or co-author of numerous publications on epidemiology and health risk assessment.

BACTERIA

303(d) Bacterial Impairments

8. Dr. Teaf states that there are 9 water bodies within the Oklahoma portion of the Illinois River Watershed (IRW) categorized as impaired for Primary Body Contact Recreation as a result of pathogens and indicator bacteria including *E. coli*, enterococci, and/or fecal coliforms per the Oklahoma Department of Environmental Quality (ODEQ 2006a) 303(d) list (para 16, Teaf 2008a).
9. ODEQ (2006a) states that the sources of the impairments for the nine water bodies are as follows:
    a. Unknown (5 water bodies)
    b. Not applicable (1 water body)
    c. Unknown and municipal discharges (1 water body)
    d. Grazing in riparian or shoreline zones, on-site treatment systems (septic systems and similarly decentralized systems), rangeland grazing, wildlife other than waterfowl, and unknown (1 water body)
    e. Grazing in riparian or shoreline zones, on-site treatment systems (septic systems and similarly decentralized systems), permitted runoff from confined animal feeding operations, rangeland grazing, total retention domestic sewage lagoons, wildlife other than waterfowl, and unknown (1 water body)
10. The ODEQ (ODEQ 2006a) does not state that the source of bacterial impairment for any of the water bodies in the IRW for 2006 is a result of poultry litter.
11. For one water body (10.3 miles of Peacheater Creek; see 9e above), ODEQ (2006a) states that one of the seven causes (including the cause "unknown") is "confined animal feeding operations" but does not specify poultry[1].
12. EPA (2008a) reported that there were 549 reported impairments because of *E. coli*, enterococci, or fecal coliform for water bodies in Oklahoma in 2006. Only 10 of these impairments occurred in the IRW (one water body was considered impaired for both enterococci and fecal coliform).

[1] Oklahoma's 2008 report was approved by EPA on October 22, 2008 (ODEQ 2008a). The 2008 report lists twelve water bodies in the IRW impaired for PBCR as the result of bacteria. Of those twelve, a source of the bacteria for three water bodies is indicated as Confined Animal Feeding Operations but not specifying poultry (ODEQ 2008a). Possible other sources for all three water bodies include on-site treatment systems (septic systems), grazing in riparian zones, rangeland grazing, wildlife other than waterfowl, and "unknown".

13. In summary, the State of Oklahoma (ODEQ 2006a) 303d list reports nine water bodies in the IRW as impaired because of bacteria but does not report that any of the impairments are the result of the application of poultry litter; a variety of potential sources for these impairments are indicated. Furthermore, bacterial impairment of recreational waters occurs all over the State of Oklahoma; it is certainly not isolated to the IRW.

Bacterial Indicators

14. Dr. Teaf reports that microbial indicator organisms are commonly used and widely accepted measures of the potential for the presence of pathogens, including bacteria, viruses, and protozoa in recreational water and cites the National Research Council (NRC 2004), EPA (2005), and Wade et al. (2006) as references (paras 17, 21 Teaf 2008a).
15. NRC (2004) states: "The use of indicators is based on the presumption that they co-occur at a constant ratio with illness-causing pathogens. This premise is flawed because indicator levels in the gastrointestinal tract may vary within a narrow range, but pathogen concentration is highly variable and dependent on which pathogens are in the population at what levels at specific times. Furthermore, upon leaving the intestinal tract, microbial indicators and pathogens degrade at different rates that are mediated by factors such as their resistance to aerobic conditions, ultraviolet radiation, temperature changes, and salinity. As a result, the epidemiological relationship between indicator density and illness patterns can differ depending on the age of the source material, as well as local meteorological and other environmental conditions. Several studies also have found that some indicator bacteria can grow outside the human or animal intestinal system, further confounding the correlation between pathogens and indicators."
16. NRC (2004) further states: "The underlying epidemiologic studies are also limited because many reported failures of beach water quality standards are associated with nonpoint source, but the epidemiologic studies used to establish recreational bathing water standards have been based primarily on exposure to human fecal-dominated point source contamination. Since nonpoint sources generally have a higher percentage of animal fecal contributions, and animals shed bacterial indicators without some of the accompanying human pathogens, there is considerable uncertainty in extrapolating present standards to nonpoint source situations. A poor correlation between bacterial indicators and virus

concentrations has been found in the study of nonpoint sources and water quality. However, when a human source, such as septic systems, has been present, enterococci have been significantly correlated with viruses."

17. EPA (2005) states: "The works of several researchers has shown that these indicators (*E. coli* and enterococci) are not reliable surrogates for many pathogens including bacteria, and most viruses and parasites. New approaches for detecting pathogens are needed to improve monitoring systems."
18. Dr. Harwood states that the link between bacterial indicators and human illness from recreational waters has been demonstrated in many epidemiological studies (para 23, Harwood 2008) and that indicator water quality standards have been supported since EPA's (1986) ambient quality criteria were published (para 26, Harwood 2008). Harwood et al. (2005), however, found that indicator organisms, including enterococci, were a poor predictor of pathogens including enteric viruses, cryptosporidium, and giardia.
19. Dr. Teaf (para 21, Teaf 2008a) claims that "These indicator organisms, such as *E. coli*, enterococci, and fecal coliform bacteria, may not cause illness directly, but they have demonstrated characteristics which make them reliable indicators of other harmful pathogens in water" and cites Wade et al. (2006) as a reference. Wade et al. (2006), however, provides no endorsement of *E. coli* or enterococci as indicator organisms and never mentions fecal coliform as an indicator organism. This was a study of a quantitative polymerase chain reaction method for evaluating enterococci and bacteroides as indicators for gastrointestinal illness. The authors stated: "Because this is the first and only study to evaluate the ability of rapid water-quality indicators to predict GI [gastrointestinal] illness, additional studies will be required to evaluate the generalizability of these findings. Additional studies and analyses will help determine whether these preliminary findings are consistent and robust enough from a regulatory perspective to recommend a rapid indicator for recreational water quality, and to evaluate the conditions under which such indicators can successfully be applied." Furthermore, the beaches studied were specifically selected because they were affected by discharges from waste treatment plants.
20. Dr. Harwood claims that acute febrile respiratory illness (AFRI) has been linked in epidemiology studies to elevated microbial pollution levels and cites Fleisher et al. (1998) as a reference (para 7, Harwood 2008). Dr. Harwood also claims that children are among

those most likely to contract gastrointestinal illness from swimming (para 9, Harwood 2008), citing Cabelli et al. (1979) and Pruss (1998) as references. Fleisher et al. (1998) reported an increased risk of AFRI associated with bathing in sewage-contaminated water. Cabelli et al. (1979) was also a study of swimmers in sewage-contaminated water. Six of the 23 studies reviewed by Pruss (1998) were fresh water studies; three of those reported the bacterial source to be sewage; the bacterial source in the other studies was not stated. These results are highly questionable in evaluating risks from non-human sources such as poultry litter, as it is widely believed that human feces pose a larger health risk than animal feces to swimmers and other primary contact recreational water users (WHO 2003).

21. In 2000, Congress passed the Beaches Environmental Assessment Act requiring the completion of studies on pathogen indicators in coastal recreational waters within 3 years and the publication of new or revised water quality criteria for pathogens and pathogen indicators within 5 years (BEACH 2000). When EPA failed to publish new Ambient Water Quality Criteria (AWQC) for bacteria in 2005, the Agency was sued by the Natural Resources Defense Council (NRDC). As part of its response to the action by the NRDC, the EPA (2007a) convened a panel of experts to obtain "input from individual members of the broad scientific and technical community on the critical path research and related science needs." The purpose of the research was to support "new or revised AWQC by 2012."

22. Dr. Harwood and Dr. Teaf both claim that EPA's expert panel (EPA 2007a) placed the highest priority for research on recreational water quality on pathogens from poultry and other agricultural animals (e.g., cattle, sheep) (para 38, Teaf 2008a; para 10, Harwood 2008). The table to which Drs. Teaf and Harwood refer in the EPA (2007a) report (Table 5) places the highest research priority on epidemiologic studies at beaches impacted by fecal contamination from "other agricultural animals (e.g., cattle, sheep)" and not beaches impacted by fecal contamination from poultry. More importantly regarding agricultural animals, EPA (2007a) states that "Current epidemiological literature suggests that the symptomatic profile of swimming-associated illnesses indicates primarily viral illnesses.....With rare exception, viruses are species-specific. Essentially, all enteric oral/fecally transmitted viruses that infect humans are of human origin." Consequently EPA (2007a) assigned a negligible risk to viruses of animal (including poultry) origin.

23. Dr. Harwood claims that WHO (2003) has adopted "standards" for water quality based on indicator bacteria (para 26, Harwood 2008). The WHO (2003) guidelines for recreational water are based on microbial water quality assessment (as measured by enterococci) and sanitary inspection. The sanitary inspection described by WHO is principally driven by human fecal inputs to the recreational water. WHO states that one of the bases of their approach is the movement away from sole reliance on fecal indicator bacteria. The approach allows a water quality assessment category to be given a more favorable category when bacterial contamination is from non-human sources.
24. Dr. Harwood states that a re-analysis by Wade et al. (2003) of 27 epidemiology studies strongly supports the relationship between indicator bacteria (*E. coli* and enterococci) concentrations and gastroenteritis rates in recreational water users. Dr. Harwood further states that enterococci are responsible for many of the water quality exceedances throughout the IRW and that this group of fecal indicator bacteria "are correlated with the risk of gastroenteritis in fresh and salt water" (para 30, Harwood 2008). Wade et al. (2003), however, found that "no increase in relative risk was observed for high levels of enterococci compared with low levels." Commenting on Wade et al. (2003), the NRC (2004) stated, "there was no best (consistent) indicator of gastrointestinal illness in freshwater."
25. Dr. Harwood concluded that indicator bacteria standards will "doubtless be used to protect the health of recreational waters in the U.S. for the foreseeable future" based on a report by EPA's expert panel (EPA 2007a). EPA (2007a) did not endorse the current AWQC for bacteria; furthermore, they recommended research on a variety of approaches, not simply limited to indicator bacteria.
26. Following the EPA (2007a) report, EPA (2007b) developed a Critical Path Science Plan to answer what the Agency considered to be the key questions in the development of a new or revised AWQC. Prior to the plan, EPA had already embarked on an extensive multibillion dollar research program, including both in-house and extramural research, to improve the science of the AWQC (Grumbles 2007, Haugland et al. 2005, Wade et al. 2006, Wade et al. 2008, EPA 2008b).
27. In summary, the validity of indicator bacteria has been challenged by expert panels formed by EPA or at the request of EPA, and it is anticipated that EPA will publish new AWQC by

2012 (NRC 2004, EPA 2007a, EPA 2007b). To this end, EPA is engaged in an extensive research program to improve the science of the AWQC, and EPA (2007b) has developed an extensive research plan to ensure that the new or revised criteria are scientifically sound.

EPA Ambient Water Quality Criteria (AWQC) for Bacteria

28. Dr. Teaf refers to the U.S. EPA (2003) *Draft Implementation Guidance for Ambient Water Quality Criteria for Bacteria*[2] (paras 21-23, Teaf 2008a). He states that these draft guidelines report that a geometric mean density of 126 *E. coli* per 100 milliliters (mL) of water and a geometric mean density of 33 enterococci per 100 mL of water over a 30-day period are associated with an illness rate of 8 illnesses per thousand recreational users.
29. The AWQC for bacteria were developed by EPA (1986) based on "acceptable risk". The determination of "acceptable risk" derives from a recommendation made by the National Technical Advisory Committee (NTAC 1968). NTAC cited studies done in the late 1940s and early 1950s at Lake Michigan and on the Ohio River in which an "epidemiologically detectable health effect" was found to occur between 2,300 and 2,400 total coliforms per 100 mL. Studies conducted on the Ohio River over a decade later estimated that 18 percent of the total coliforms were fecal coliforms (i.e. about 400 fecal coliforms per 100 mL). The NTAC (1968) recommended fecal coliforms as a better indicator of disease than total coliforms and recommended using a safety factor of two, resulting in a Water Quality Criteria of 200 fecal coliforms/100 mL for primary recreational waters as an acceptable risk.
30. The epidemiologic studies which became the basis of the AWQC were conducted at beaches at Lake Erie, Pennsylvania and Keystone Lake, Oklahoma between 1979 and 1982. Two beaches were studied at each location. At both locations, the source of the bacteria was treated sewage. Persons studied were swimmers and nonswimmers at the two beaches. Swimming activity was rigidly defined as having all upper body orifices exposed to the water. The determination of illness was based on telephone interviews conducted 8 to 10 days after the individuals swam at the beach. A statistically significant increase in "highly credible gastrointestinal symptoms (HCGI)" was found only at the beach with

[2] The Final Implementation Guidance for Ambient Water Quality Criteria for Bacteria was published in 2004 (EPA 2004b).

poorer water quality at Lake Erie and was not found at either of the two beaches studied at Keystone Lake (EPA 1984). The symptom category HCGI was defined as including any one of the following symptoms: (1) vomiting, (2) diarrhea with fever or a disabling condition (remained home, remained in bed or sought medical advice because of the symptoms) and (3) stomachache or nausea accompanied by a fever. EPA used this category to define gastrointestinal illness rather than total GI symptoms because, as the Agency acknowledged, all of the symptoms were self-diagnosed and therefore subject to variable interpretation.

29. Regression equations of the geometric means of enterococci and *E. coli* densities per 100 mL versus HCGI were developed to estimate the densities that would be associated with the "acceptable risk" of 200 fecal coliforms/100 mL[3]. These densities were estimated to be 126 and 33 per 100 mL for *E.Coli* and enterococci, respectively. EPA (1986) stated that "while this level is based on the historically accepted risk, it is still arbitrary insofar as the historical risk itself was arbitrary."
30. The "acceptable risk" for gastrointestinal illness was estimated to be 8 per 1,000. EPA (1986) described this estimated risk as "only approximate".
31. EPA (1986) stated that "based on a statistically significant number of samples (generally not less than 5 samples equally spaced over a 30 day period)", water samples should not exceed these concentrations of *E.coli* and enterococci. Furthermore, EPA stated that "In deciding whether a beach should be left open, it is the long term geometric mean bacterial density that is of interest. Because of day-to-day fluctuations around this mean, a decision based on a single sample (or even several samples) may be erroneous, i.e., the sample may exceed the recommended mean criteria even though the long-term geometric mean is protective, or may fall below the maximum even if this mean is in the nonprotective range." The National Research Council (NRC 2004) stated that "it is clear that little information can be obtained from analysis of a single sample of water for a microbial indicator or pathogen."

[3] Although EPA (1986) used the "acceptable risk" based on fecal coliform data from the 1968 recommendations, it did not recommend the use of fecal coliform as an ambient water criterion because it was noted that in some cases, fecal coliforms are routinely detected where fecal contamination is absent.

32. EPA (2007a) stated that "It is not certain how accurate the current levels of protection are. 'Magic' numbers like 8 or 19 cases of gastroenteritis in 1,000 swimmers can 'take on a life of their own,' increasing the risk of distraction from the basic objective—providing best effort to protect swimmers. This provides a compelling reason for not deriving and using a single numeric value for the targeted risk for new or revised AWQC."
33. The fecal contamination at Keystone Lake and Lake Erie, studied by EPA to derive the AWQC, was of human origin. The WHO (2003) stated that the use of fecal bacteria alone as an index of risk to human health may significantly overestimate risks where the index organisms derive from sources other than human excreta. EPA (2007a) stated that it is widely believed that human feces pose a larger health risk than animal feces to swimmers and other primary contact recreational water users. This belief derives from the basic concept that virtually all enteric pathogens of humans are infectious to other humans, while relatively few of the enteric pathogens of animals are infectious to humans. A study by Calderon (1991) found no evidence of risk to swimmers in a water body heavily contaminated by unidentified sources of animal feces. A study by Colford et al. (2007) found no evidence between levels of traditional fecal indicator bacteria and illness in Mission Bay, California, where the predominant source of fecal contamination was avian. The authors believed their results to be due to a lack of human sources of traditional fecal indicator bacteria.
34. The recreational activities described by Dr. Caneday suggest that the majority of recreational activity in the IRW does not involve having all upper body orifices exposed to water (Caneday 2008). Dr. Caneday describes the primary recreational activity as "floating" in which he includes the use of canoes, kayaks, rafts and other inflatable floats, but indicates that both "floaters" and "nonfloaters" will swim in the river.
35. Dr. Dunford (2008), however, states that almost all water contact of the floaters in the IRW is dermal and full immersion is infrequent and short-lived. Extended periods of immersion are not possible in most places because the river is too shallow. "In general the Illinois River is a floating river, not a swimming river" (para 16, Dunford 2008).
36. The primary activities which Drs. Caneday and Dunford describe fall under EPA's definition of secondary contact. EPA (2004b) defines secondary contact uses as "including activities where most participants would have very little direct contact with the water and

where ingestion of water is unlikely. Secondary contact activities may include wading, canoeing, motor boating, fishing, etc." EPA (2004b) further states, "Adoption of a secondary contact criterion is also consistent with historical practices for most states and authorized tribes. Accordingly, states and authorized tribes may wish to adopt a secondary contact criterion which is five times their primary contact criterion." In other words, EPA would allow the water quality criteria to be five times higher if the recreational activities were wading, canoeing, motor boating, fishing, etc.

37. Dr. Teaf (para 24, Teaf 2008a) describes primary body contact recreation as "involving direct body contact (i.e., the dermal exposure route)." Again, the AWQC for Bacteria were developed from epidemiologic studies in which swimming activity was rigidly defined as having all upper body orifices exposed to the water, indicating exposure via ingestion as opposed to dermal contact alone.
38. Dr. Teaf states that for bacterial levels above the AWQC the "Agency (EPA) noted that the illness rates rise sharply" (para 22, Teaf 2008a) and that "the epidemiological data illustrate an exponential increase in rates beyond the 1.0% illness incidence threshold, instead of a linear increase." What Dr. Teaf is referring to as an "exponential increase in rates beyond the 1.0% illness rate" is derived from a conceptual model (EPA 2004b). There are no epidemiologic data to determine whether this relationship exists or not.
39. Dr. Teaf (2008) frequently makes use of the term, "illness rate". In a clarification of the term "illness rate", EPA (2004b) explains "There has been confusion surrounding the use of several terms related to EPA's 1986 bacteria criteria. First, the use of the term 'illness rate' implied a precision in predicting risk that current data do not support. There is a certain degree of uncertainty and variability associated with illness rates and indicator densities, and the term 'risk level' better captures the true meaning of the concept."
40. Dr. Teaf states that it is possible to estimate that over 1,200 illnesses are occurring on an annual basis if the bacterial water quality is just being met in the IRW (para 23, Teaf 2008a). Dr. Teaf (2008b) agreed in deposition, however, that no individual in the IRW had been identified with illness associated with recreational water contact. The Centers for Disease Control and Prevention (CDC) periodically reports on waterborne-disease outbreaks associated with recreational water (Barwick et al. 2000, Dziuban et al. 2006, Yoder et al. 2004, Lee et al. 2002, Kramer et al. 1996, Levy et al. 1998, Moore et al.

1993). There were no reports of bacterial waterborne disease outbreaks associated with untreated recreational waters in the entire state of Oklahoma for the years 1991-2004.

41. In summary, the relationship of the AWQC to bacteria from non-human sources has been challenged by the NRC (2004) and by EPA (2007a). EPA intends to set new or revised AWQC by 2012. AWQC based on studies of swimmers in which it was required that the swimmers' heads be immersed during activities were not intended to be applied to wading, canoeing, fishing, and "floating". Dr. Teaf cites a *conceptual* model for which there is no epidemiologic evidence to claim that there is an exponential increase in risk above the AWQC for bacteria. There have been no reports of disease associated with recreational water activities occurring in the IRW reported to the CDC for the last ten years.

Total Maximum Daily Load (TMDL)

42. Dr. Teaf has presented an ad hoc TMDL analysis (paras 30-32, Teaf 2008a) and uses EPA (2001) as guidance. EPA (2001) "strongly recommends" not using fecal coliform as a bacterial indicator; nevertheless, Dr. Teaf, uses fecal coliform as the indicator for his analysis. EPA (2001) also stated "It is also important to realize that the presence of indicator bacteria does not always prove or disprove the presence of human pathogenic bacteria, viruses, or protozoans."
43. There is no evidence that Dr. Teaf has been asked to prepare a TMDL for the State of Oklahoma. A TMDL requires, among other things, public participation, and there is detailed guidance for TMDL development, which Dr. Teaf has followed to only a limited extent. One of Dr. Teaf's reliance documents is ODEQ (undated). ODEQ (undated) states that they have the statutory authority to lead the development of TMDLs, but that EPA's stringent review and approval procedures must be met before any TMDL can be implemented. It also states that a rationale for selecting a specific water quality model should be provided. ODEQ (undated) states that watershed models typically require a wider variety of data sources than near field models because they cover a broader geographic area and account for more transport mechanisms such as stormwater runoff and atmospheric deposition.
44. In summary, Dr. Teaf has not followed the guidance of either the EPA or the ODEQ in developing his ad hoc TMDL.

Pathogens in Poultry Litter

45. Dr. Teaf states that bacteria of human health significance, including *Campylobacter*, *Salmonella*, *Staphylococcus*, *Escherichia coli* and other important species, are present in poultry litter and cites Kelley et al. (1995), Jenkins et al. (2006), and Pew Commission on Industrial Farm Animal Production (PCIFAP 2008) as references. None of the authors of these references studied *Staphylococcus* in poultry litter. Jenkins et al. (2006) studied both *Salmonella* and *Campylobacter* and found neither in poultry litter. The PCIFAP report makes no mention of pathogens in poultry litter. The Kelley et al. (1995) study didn't find *Salmonella* in poultry litter.
46. Dr. Teaf claims that poultry litter is a source of giardiasis and cryptosporidiosis (para 38, Teaf 2008a). None of the references which he cites, however, support this contention.
47. *Giardia* and *Cryptosporidium* are protozoans. In evaluating disease risk in recreational water due to different pathogen sources, EPA (2007a) assigned a negligible risk to protozoa from poultry. By comparison, EPA (2007a) assigned a high risk from protozoa due to fecal shedding from child bathers, a high risk to sewage, a medium risk from "other agricultural animals (e.g., cattle, sheep)", a medium risk from "other wildlife (e.g., deer)", a low risk from aquatic birds, a low risk from fecal shedding from adult bathers, and a low risk from domestic animals (e.g., dogs, cats).
48. In summary, Dr. Teaf's references do not support his claims of pathogens in poultry litter.

Salmonellosis and campylobacteriosis

49. Dr. Teaf states that Oklahoma State Department of Health (OSDH) records show that Adair County reported rates of campylobacteriosis that are considerably in excess of the state average for the period from 1997 to 2005 and rates of salmonellosis that periodically exceeded the average statewide average rate for the years 1990-2005 (para 39, Teaf 2008a). He also states that the rate of salmonellosis in Sequoyah County was reported to exceed the State rate for all but three years during the period from 1990 to 2001. In Appendices 1-10, the rates of campylobacteriosis and salmonellosis are graphed for all the counties in the state for the years 2002-2007 (OSDH 2008a, 2007a, 2006a, 2005a, 2004a, 2003). The counties in the IRW are color-coded to distinguish them from the rest of the counties.

These charts illustrate that there are many counties with higher rates of campylobacteriosis and salmonellosis than either Adair or Sequoyah County for each of the years, and there is no temporal trend with regard to yearly rates for either Adair or Sequoyah County or for any county in the IRW. Furthermore, there are counties in the IRW that have lower rates than the State of Oklahoma average for the years 2002-2007.

50. The rates of salmonellosis and campylobacteriosis for Adair and Sequoyah Counties are not specific for waterborne transmission. Although *Salmonella* and *Campylobacter* can be transmitted through water, most cases are believed to be transmitted through food (APHA 2004; WHO 2005; WHO 2000). Mead et al. (1999) state that 95% of salmonellosis cases and 80% of campylobacteriosis cases are foodborne. Even the OSDH states that most cases of salmonellosis and campylobacteriosis are caused by eating contaminated food (OSDH 2007b; OSDH 2005b). CDC reports that most cases of campylobacteriosis are associated with handling raw poultry or eating raw or undercooked poultry meat (CDC 2005) and that *Salmonella* are usually transmitted to humans through ingestion of foods contaminated with animal feces (CDC 2006).
51. There are multiple reservoirs for both *Salmonella* and *Campylobacter*. The American Public Health Association (APHA 2004) states that reservoirs of *Campylobacter* include poultry, cattle, puppies, kittens, other pets, swine, sheep, rodents and birds. Reservoirs of *Salmonella* include domestic and wild animals (e.g., poultry, swine, cattle, rodents), pets (e.g., iguanas, tortoises, turtles, terrapins, chicks, dogs and cats), and humans.
52. The OSDH web site (OSDH 2008a, 2007a, 2006a, 2005a, 2004a, 2003) reports investigations of salmonellosis and campylobacteriosis outbreaks for the years 2002-2007. In that period of time, there were seven outbreaks of salmonellosis and four outbreaks of campylobacteriosis investigated. None were found to be associated with drinking or recreational water.
53. Dr. Harwood (para 18, Harwood 2008) describes two studies of waterborne salmonellosis (Angulo et al 1997; O'Reilly et al. 2007) but does not describe the source of the contaminated water in either study. The outbreak studied by Angulo et al. (1997) was of a community where the drinking water supply was exposed to outside contamination and was not chlorinated. O'Reilly et al. (2007) found that sewage contamination of ground water was the likely cause of the outbreak which they investigated.

54. Dr. Teaf and Dr. Harwood claim there is underreporting of waterborne enteric disease cases (paras 40-41, Teaf 2008a; paras 8, 15, 16; Harwood 2008). If there is underreporting of waterborne disease, there is also underreporting of foodborne disease. As noted previously, Mead et al. (1999) estimated that 95% of salmonellosis and 80% of campylobacteriosis cases are foodborne. The issue of underreporting is not limited to waterborne exposure or to the IRW and does not suggest a greater problem in the IRW than in any other area of Oklahoma or the U.S.
55. Dr. Harwood states that Friedman et al. (2004) found that drinking untreated water from a river, lake or stream is a known risk factor for campylobacteriosis (para 12, Harwood 2008). Friedman et al. (2004) examined a number of risk factors for campylobacteriosis, including factors related to dining location, food consumed, kitchen and food-handling practices, demographic characteristics, animal exposure, contact with animals with diarrhea, water exposure, and other exposure. Friedman et al. (2004) found that the greatest percentage of cases was attributable to eating chicken at a restaurant (24%) followed by eating nonpoultry meat prepared at a restaurant (21%). Having contact with an animal stool, having a pet puppy, having contact with farm animals (for persons $\geq$ 12 years of age), and eating turkey at a restaurant all accounted for more cases than drinking untreated water from a river, lake, or stream (which accounted for only 3% of cases).
56. Dr. Harwood cites Allos (2001) as evidence that contaminated water is a known source of campylobacter infections (para 12, Harwood 2008). Allos (2001) states that "The single most important route of *Campylobacter* infections in the United States and other industrialized nations remains the consumption and handling of chicken" and that other causes of sporadic cases are consumption of sausages or red meat, water, contact with pets and international travel.
57. Dr. Harwood claims that the prevalence of *Salmonella* is higher in poultry feces than in cattle feces and cites Hutchison et al. (2004) as her reference (para 13, Harwood 2008). Hutchison et al. (2004) did find that the percentage of poultry litter samples containing *Salmonella* (17.9 percent of fresh samples, 11.5 percent of stored samples) was higher than the percentage of cattle waste samples containing *Salmonella* (7.7 percent for fresh samples, 10 percent for stored samples). The authors also found, however, that the level of *Salmonella* in the cattle samples was 10 times higher than in the poultry samples.

58. Dr. Harwood reports that one drop of blood from a poultry carcass contaminated by feces can contain up to 500 infectious *Campylobacter jejeuni cells* and cites Hood et al. (1988) as a reference (para 13, Harwood 2008). Hood et al. (1988) did not report on the number of infectious cells in a drop of blood from a poultry carcass. More importantly, Hood et al. (1988) studied chickens prepared for consumption, which is not related to the issue of *Campylobacter* in chicken litter. Consumption of chicken is a well-known route of exposure for *Campylobacter* (Allos 2001; Friedman et al. 2004; Mead et al. 1999).
59. In summary, Dr. Teaf's use of campylobacteriosis and salmonellosis rates for counties in the IRW to claim that rates of these diseases are elevated because of contaminated water ignores the fact that the vast majority of salmonellosis and campylobacteriosis is transmitted by food. Furthermore, the OSDH has reported no cases of waterborne salmonellosis or campylobacteriosis for 2002-2007. The underreporting of cases described by Drs. Teaf and Harwood does not suggest a causal association with poultry litter. Dr. Harwood cites references indicating that salmonellosis and campylobacteriosis have been waterborne, but ignores the fact that the authors whom she references conclude that the vast majority of these diseases are foodborne.

ISSUES OTHER THAN BACTERIA

Disinfection By-products (DBP)

60. Dr. Teaf (para 48, Teaf 2008a) describes several epidemiologic studies which he indicates demonstrate a causal association between total trihalomethanes (TTHM) and bladder and rectal cancer. The most recent international review of the epidemiologic literature on the subject was published by WHO (2000) which stated: "The existing epidemiological data are insufficient to allow a conclusion that the observed associations between bladder or any other cancer and chlorinated drinking-water or THMs are causal or provide an accurate estimate of the magnitude of risk." Dr. Teaf also asserts (para 48, Teaf 2008a) that the National Toxicology Program (NTP), International Agency for Research on Cancer (IARC), and the U.S. EPA have concluded that the weight of evidence from animal and human studies warrants classification of the THMs, as well as at least two of the haloacetic acids (HAA5), as probable or possible carcinogens. These organizations reviewed both human and animal studies, but the classifications to which Dr. Teaf refers are all based on

laboratory animal data. The human evidence is considered inadequate for determining if THMs or HAA5s have carcinogenic activity.

61. Dr. Teaf reports concentrations of dibromochloromethane, bromodichloromethane, bromoform, and dichloroacetic acid estimated by EPA to be associated with a 1 in a 1,000,000 cancer risk (para 50, Teaf 2008a). These concentrations were estimated from rodent (mice and rat) studies in which the lowest doses that the animals received were equivalent to concentrations at least 10,000 times greater than the EPA estimates for a 1 in a 1,000,000 risk (i.e., there is no direct evidence, even in animals, that a risk of 1 in a 1,000,000 exists at the concentrations reported by EPA and cited by Dr. Teaf). Dr. Teaf fails to mention that EPA's Integrated Risk Information System (IRIS) also reports the concentrations of these chemicals associated with risks of 1 in 100,000 and 1 in 10,000; Dr. Teaf only makes comparisons to the concentration associated with a 1 in a 1,000,000 risk. As the Associate Director for Health at the National Center for Environmental Assessment at EPA, I had responsibility for EPA's (2008b) IRIS program. The concentrations of substances in water or air that are associated with risks of 1 in a 1,000,000, 1 in 100,000, and 1 in 10,000 are given for information purposes. EPA does not regulate to risks of 1 in a 1,000,000 as implied by Dr. Teaf's statement that EPA has a "standard regulatory benchmark" of a 1 in a 1,000,000 risk. A comparison of IRIS quantitative cancer risk estimates (EPA 2008c) to drinking water Maximum Contaminant Levels (MCL) (EPA 2008d) confirms that MCLs are not equivalent to concentrations representing a 1 in a 1,000,000 risk.

62. Dr. Teaf reports that Wang et al. (2007) have shown that water quality standards for the THMs need to be reviewed to account for the different risks resulting from each individual THM species. Wang et al.'s (2007) conclusions were based on theoretical calculations using the EPA cancer slope factors for the THMs and exposure modeling. These calculations were then used to estimate cancer risks for different cities in Taiwan. Wang et al. (2007) concluded that "Due to the variations of the THMs compositions, it is observed that higher concentrations of total THMs do not necessarily lead to higher cancer risks. Areas with higher bromide concentration in raw water and often with higher total THM concentration may actually give lower cancer risk if the THMs formed shift to bromoform." The authors further state "Although lower risk is expected, the presence of

higher bromoform concentration in water may lead to the violation of THM standards since bromoform has much higher molecular weight than chloroform."

63. Dr. Teaf also reports that the U.S. EPA Region 6 risk-based screening level for chloroform in residential water is 0.17 µg/L. He neglects to tell the reader that this is a conservative screening value whose purpose is to: screen residential and industrial sites to determine further evaluation, prioritize multiple sites within a facility, and focus future risk assessment efforts (EPA 2007c). The Region 6 screening level is over 400 times less than EPA's drinking water Maximum Contaminant Level Goal (MCLG). The definition of the MCLG is "the level of a contaminant in drinking water below which there is no known or expected risk to health. MCLGs allow for a margin of safety and are non-enforceable public health goals." (EPA 2008d).
64. Dr. Teaf states that there were "exceedances" and "near exceedances" of the MCLs for TTHM and HAA5, MCLGs, "risk-based values" and the Region 6 screening level value in samples taken from 18 public water systems which draw water from the IRW (paras 51-52, Teaf 2008a). Dr. Teaf's evaluation is based on single samples, not the monitoring evaluation required by EPA. Concentrations above the MCLG, Region 6 screening levels, or "risk-based values" are not drinking water violations, however. Only when the monitoring defined by EPA[4] exceeds the MCL is the concentration considered a violation.
65. Dr. Teaf claims, that based on ODEQ data for early 2008, there were 56 "exceedances" of the total trihalomethanes (TTHM) MCL and 35 "exceedances" of the haloacetic acids (HAA5) MCL for the 18 active drinking water systems serving the IRW in 2008 (i.e., a total of 91 "exceedances" for the DBPs which have MCLs) (para 51, Teaf 2008a). The State of Oklahoma reports that among those 18 water treatment systems described by Dr. Teaf, there was one with DBP violation(s) in 2007, none in 2006, five in 2005, and two in 2004 (ODEQ 2008b, 2007, 2006b, 2005).

[4] For surface water systems serving more than 10,000, EPA monitoring compliance for the MCLs for TTHMs and HAA5 is based on a running annual average of quarterly samples in which at least 4 samples are taken per quarter. For surface water systems serving less than 10,000, compliance for the MCL is based on a running annual average of at least one sample per quarter. For ground water systems serving greater than 10,000 population, the MCL is based on a running annual average of at least one sample taken per quarter. For groundwater systems serving less than 10,000, compliance is based on one sample per year. If that annual monitoring result exceeds the MCL, the system must increase monitoring frequency to 1/plant/quarter, and compliance determinations will be based on the running annual average of quarterly monitoring results (EPA 1998).

66. Using Plaintiff sponsored, Camp, Dresser, and McKee (CDM) data, Dr. Teaf describes 6 "exceedances" of the TTHM MCL and three "exceedances" of the HAA5 MCL for three IRW public water systems - Tahlequah, Gore PWA, and Cherokee RD#2 (para 52, Teaf 2008a). However, the State of Oklahoma reports that there were no DBP violations for either Cherokee RD#2 or Tahlequah for the years 2004-2007 and Gore PWA was only in violation in 2005 (ODEQ 2008b, 2007, 2006b, 2005).
67. Dr. Teaf indicates that elevated levels of THMs and HAA5 "often result in esthetic concerns", such as undesirable taste and odor, but provides no evidence of esthetic problems occurring in the IRW.
68. There is no evidence that DBP violations occur to any greater extent in the IRW than any other place in Oklahoma. Percentages of drinking water systems in violation of the MCLs for DBPs are presented for all counties in Oklahoma for the years 2004-2007 in Figure 13. As illustrated, DBP violations in the counties of the IRW are among the lowest across the state. When the number of systems having DBP violations are presented by zip code (Figures 14), the same pattern emerges (ODEQ 2008b, 2008c, 2007, 2006b, 2005).
69. In summary, Dr. Teaf has misrepresented the evidence on the health effects of DBPs and made improper comparisons with respect to the MCLs for the DBPs. He has made comparisons to MCLGs and to a screening value; drinking water violations are determined by evaluation of drinking water concentrations vis-à-vis the MCL, not MCLGs and screening values. Finally, while DBP drinking water violations have occurred in the IRW, they occur to a lesser extent than in other parts of Oklahoma.

Cyanobacteria

70. Cyanobacteria are organisms with some characteristics of algae and some of bacteria. They are similar to algae in size, and unlike some bacteria, they contain blue-green and green pigments and can perform photosynthesis. Worldwide, about 60% of cyanobacteria contain toxins (i.e., not all cyanobacteria are toxic). There are a variety of toxins found in cyanobacteria. Microcystins (produced by the cyanobacteria species, *Microcystis*) are the most frequently occurring and widespread of the cyanotoxins (WHO 2003).
71. WHO (2003) guidelines indicate that there is a low probability of adverse health effects if the cyanobacterial density is less than 20,000 cyanobacterial cells/mL or there is less than

10 µg chlorophyll-a/L with dominance of cyanobacteria. A moderate probability of adverse health effects is said to exist if the cyanobacterial density is less than 100,000 cells/mL or 50 µg chlorophyll-a/L with dominance of cyanobacteria. A high probability of adverse health effects is said to exist if there is scum formation in areas where whole-body contact or ingestion/aspiration could potentially occur.

72. Dr. Teaf claims that sampling in Lake Tenkiller during August 2004 through August 2007 show that 58% of all samples had cyanobacterial densities greater than 20,000 cells/mL, 24% of samples had cyanobacterial densities greater than 100,000 cells/mL, and one sample exceeded 1,000,000 cells/mL (para 63, Teaf 2008a).
73. Cyanobacteria are ubiquitous in nature and found in nearly all environments. For example, the United States Geological Survey (USGS 2004) reported that the average cell count of cyanobacteria for one site at Fort Cobb Reservoir in Caddo County in western Oklahoma was >200,000 cells/mL and greater than >100,000 cells/mL for two sites. For two other sites, the average cell counts were >50,000 cells/mL, and for two others, the average cell counts were >20,000 cells/mL.
74. The Oklahoma Water Resources Board (OWRB 2004) found that the algae cell density in Lake Thunderbird in central Oklahoma was predominated by cyanobacteria. Average algal cell density was greater than 100,000 cells/mL for three different years (2001, 2002, and 2003) and almost 1,000,000 cells/mL in 2001. Cyanobacteria constituted greater than 90% of this density.
75. WHO (2003) states that if microcystin-producing bacteria are dominant, 2 to 4 µg microcystin/L may be expected when the cyanobacteria cell density is 20,000 cells/mL (WHO's guidance level of a low probability of adverse health effects). At 100,000 cyanobacteria cells/mL (WHO's guidance level of a moderate probability of adverse health effects) a concentration of 20 µg microcystin/L may be expected if the cyanobacterial bloom is *Microcystis*.
76. Lynch and Clyde (2006) reported microcystin concentrations of 0.35 and 3.3 µg/L, but no detection of cylindrospermopsin (another cyanotoxin), in Lake Tenkiller at two sites (pg 32, Cooke and Welch 2008). CDM did not detect any microcystin in the samples collected at Lake Tenkiller (pg 32, Cooke and Welch 2008). In other words, the microcystin

concentrations in Lake Tenkiller would suggest low probability of health effects according to the WHO guidelines.

77. Like other cyanobacteria, *Microcystis* is ubiquitous and has been found in lakes throughout the United States, especially in the upper Midwest. Thirty-two percent of lakes sampled throughout the U.S. contained microcystin (USGS 2008). See Figure 15.
78. Between 1999 and 2006, seventy-eight percent of lakes in the upper Midwest (Missouri, Iowa, parts of Kansas and Minnesota) had at least one occurrence of toxins. Concentrations of microcystin ranged from <0.10 to 52 µg/L during this period of time (USGS 2008). In a 2006 study of the upper Midwest, microcystins were the dominant class of cytotoxins detected. In 23 lakes studied, microcystin was found in 96% of samples compared to 30% for anatoxin-a and 9% for nodularin.
79. Lynch and Clyde (2006) reported that Lake Marion in Kansas had a maximum level of 16.4 µg/L microcystin and Fort Gibson in Oklahoma had a maximum 4.4 µg/L microcystin.
80. Cyanobacteria have been studied extensively in the Great Lakes and Lake Champlain. The western basin of Lake Erie is characterized by high biomass blooms of *Microcystis aeruginosa* that produced microcystins at concentrations exceeding 20 µg/L. In Missiquoi Bay in Lake Champlain, average shoreline concentrations of microcystins taken on four different dates: August 19, 2003, August 24, 2003, July 26, 2004, and September 1, 2004 were 6.2, 23.9, 78.3, and 6490 µg/L, respectively (Great Lakes Research Consortium 2006).
81. EPA has not issued guidance on either cyanobacteria or microcystin in recreational or drinking water. While some states have issued guidance on cyanobacteria, Oklahoma is not one of them. Nebraska, for example, has a Health Alert Declaration of 20 parts per billion (20 µg/L) for the cyanotoxin, microcystin. As of September 7, 2008, five lakes in the state were under environmental alert, with microcystin levels ranging from 21.34 to73.15 µg/L. Lakes under the Health Alert are sampled weekly, and the Health Alert stays in effect until the level stays below 20 µg/L for two consecutive weeks (NDEQ 2008).
82. Cooke and Welch report levels of chlorophyll in excess of the state's water quality standard of 10 µg/L in Lake Tenkiller, with summer averages up to 30 µg/L (pg 25, appendix D, Cooke and Welch 2008). Dr Teaf reports long term averages of 6.4, 8.7, 15.9

and 27.1 µg/L of chlorophyll-a in Lake Tenkiller (Table C1, Teaf 2008a). To select an appropriate reference lake, Dr. Olsen describes summer average chlorophyll-a concentrations (µg/L) for other water bodies: Stockton Lake 5.7; Clearwater Lake 4.5; Table Rock 4.3 (Table 2.13-3, Olsen 2008). In his summary tables, Dr. Olsen reports average measures of 11.4 µg/L, corrected, and 12.9 µg/L, uncorrected, for chlorophyll-a in Lake Tenkiller; 4.7 µg/L, corrected, and 5.4 µg/L, uncorrected, for Broken Bow; and 18.3 µg/L, corrected, and 21 µg/L for Stockton Lake (Appendix C, Tables 9, 15, 16, Olsen 2008). All three reports (Cooke and Welch 2008; Olsen 2008; and Teaf 2008a) claim that elevated chlorophyll-a values indicate increased health risks from exposures to cyanobacteria toxins in Lake Tenkiller.

83. In contrast to the data cited by Drs. Teaf, Olsen, Cooke and Welch, data bases provided by the plaintiffs show somewhat different results with respect to chlorophyll-a. The USGS (2006) reported that the average chlorophyll-a for 210 samples collected at Lake Tenkiller for the period of 1996-2003 was 1.55 µg/L, and only one of the samples was above 10 µg/L; two were 10 µg/L. The OWRB (2002) reported that chlorophyll-*a* concentrations in Lake Tenkiller ranged from 2.18 to 38.94 µg/L (n=23) with an average of 15.66 µg/L and a median of 11.9 µg/L (OWRB 2002).

84. Data not cited by the State but reported by OWRB and USGS suggest that other lakes in Oklahoma have a considerably greater problem with chlorophyll-a than does Lake Tenkiller. 71% of samples at Lake Thunderbird in central Oklahoma had chlorophyll-a concentrations >10 µg/L, and all 2-year rolling averages between 2002 and 2006 were above 10 µg/L (OWRB 2007). At Fort Cobb Lake in western Oklahoma, concentrations of chlorophyll-a measured at 18 different sites over a two year study period (June 2000 through June 2002) ranged from 0 to 250 µg/L, while the average chlorophyll-a concentrations ranged from 21 to 83 µg/L (USGS 2004).

85. The OSDH (OSDH 2007c, 2006b, 2005c, 2004b) Epidemiology Bulletins make no mention of any outbreak with respect to cyanobacteria associated with non-treated recreational water. The OSDH did not issue any press releases in 2004-2008 warning of adverse health effects or detection of disease associated with cyanobacteria (OSDH 2008b, OSDH 2007d, OSDH 2006c, OSDH 2005d, OSDH 2004c). The CDC has not reported any disease outbreaks due to cyanobacteria in Oklahoma for the years 1991-2004 (Moore et al.

1993, Kramer et al. 1996, Levy et al. 1998, Barwick et al. 2000, Lee et al. 2002, Yoder et al. 2004, Dziuban et al. 2006).

86. In summary, cyanobacterial cell density, microcystin levels, and chlorophyll-a concentrations in Lake Tenkiller are similar to those found in lakes in Oklahoma that are not in the IRW. In fact, there are lakes in Oklahoma and the rest of the U.S. with considerably more cyanobacteria problems than Lake Tenkiller. Microcystin concentrations in Lake Tenkiller indicate that there is a low probability of health effects per the WHO guidelines. The State of Oklahoma has issued no press releases or epidemiology bulletins on cyanobacteria. Although there have been reports of cyanobacteria-related illness in states other than Oklahoma (e.g., 22 cases in Nebraska in 2004 (Dziuban et al. 2006)), the CDC has not reported any outbreaks of cyanobacteria-related illness in Oklahoma for the last 10 years.

Nitrates, Metals, and Hormones

87. Dr. Fisher, a consultant for the State, states that the only contaminants of concern in the IRW are phosphorus and bacteria (pg 451, 516, Fisher 2008) and specifically states that there are no problems due to nitrates, metals or hormones (pg 615-616, Fisher 2008). Furthermore none of the State's consultants have raised nitrates, arsenic, copper, zinc, or hormones as health issues. These contaminants have been identified by State's consultantss and therefore are discussed below with regard to whether they could be related to poultry litter and any health implications they may present.

Nitrates

88. Mr. King reports that 8 of 60 private wells sampled by CDM in 2006 and 2007 were reported to have total nitrogen (N) results greater than 10 mg N per liter (L) and claims that this exceeds the MCL for nitrate (para 2.3.3, King 2008). Mr. King does not, however, indicate that nitrate in ground water is a human health risk in the IRW (para 2.1, King 2008).

89. The USGS (1998) evaluated water quality in the Ozark plateaus in 1992-1995. The Ozark plateaus include parts of Arkansas, Kansas, Missouri, and Oklahoma. One plateau, the Springfield Plateau, includes the IRW. It was reported that very few ground water samples

(< 1%) and no surface water samples in the Ozark plateaus exceeded the MCL for nitrate (10 mg/L measured as N). The median nitrate concentrations in Springfield Plateau aquifer springs and wells were about 2.6 and 1.0 mg/L, respectively, posing little or no health risk.

90. Drinking water MCLs apply to public water systems, not to the private wells sampled by CDM. Furthermore, 8 of 60 wells (13%) with concentrations above the MCL for nitrate is not unusual for private wells in agricultural areas. Ward et al. (2005) reported that about 22% of domestic wells in agricultural areas of the United States contain nitrate levels that exceed the MCL. About 10% of Wisconsin's 800,000 private wells have nitrate-nitrogen concentrations exceeding the MCL, and in agricultural areas the percent exceeding the MCL is between 17% and 26%. In one agricultural area in Wisconsin (Stevens Point), the percent of wells with nitrate exceeding the MCL was reported to be over 60%. Fifty-four of the 1,000 wells (5.4%) used by schools, churches and businesses on an everyday basis in Wisconsin were reported to have nitrate levels greater than the MCL (The Nutrient Management Subcommittee of the Nonpoint Source Pollution Abatement Program Redesign 1999). A survey of private well-water users in Iowa found that 35% of wells less than 50 feet deep had nitrate levels exceeding the MCL (Kross et al. 1993).

91. In summary, Mr. King has made no claims of health effects occurring in the IRW as a result of nitrate in drinking water. The MCL for nitrate applies to public water systems, not to the private wells for which Mr. King claims that the MCL is exceeded. In contrast to the CDM data cited by Mr. King, the USGS found no evidence of a nitrate problem in wells in the IRW in the 1992-1995 time period. Regardless, nitrate occurrence in agricultural areas is not uncommon. It is estimated that 22% of domestic wells in agricultural areas of the U.S. exceed the MCL for nitrate compared to the 13% which Mr. King claims exceed the MCL in the IRW based on CDM data.

Arsenic, Copper, and Zinc

92. Dr. Olsen (Tables 6.5-1 and 6.5-2, Olsen 2008) reported that copper, zinc, and arsenic were detected in Lake Tenkiller, springs and ground water in the IRW. He did not indicate that those metals presented any health concerns. The average concentrations of arsenic in Lake Tenkiller, springs, and groundwater reported by Dr. Olsen were 2.7, 2.5, and 0.8 µg/L, respectively. The drinking water MCL for arsenic is 10 µg/L. The average concentrations

of copper in Lake Tenkiller, springs, and groundwater in the IRW reported by Dr. Olsen, are 1.4, 1.6, and 19.5 µg/L, respectively. The MCL for copper is 1300 µg/L and no more than 10% of tap water samples may exceed this value. The average concentrations of zinc in Lake Tenkiller, springs, and groundwater in the IRW reported by Dr. Olsen, are 4.1, 4.6, and 37 µg/L, respectively. The secondary standard[5] for zinc in drinking water is 5,000 µg/L.

93. In summary, Dr. Olsen claims no health effects from zinc, copper, or arsenic in the IRW. That is consistent with the data which he presents showing concentrations of these elements below the MCL, action level, or secondary standard.

Hormones

94. Dr. Olsen reports estrone concentrations in Lake Tenkiller, springs, and groundwater to be 1.27, 0.645, and 0.5 ng/L (Tables 6.5-1 and 6.5-2, Olsen 2008). All animals, including humans, excrete hormones. Wang et al. (2008) reported that, hormone excretion in women is affected by the phase of the menstrual cycle and pregnancy status. Pregnant women may excrete up to 30 mg of estrogen (mainly estriol) daily.
95. Lishman et al. (2006) reported that the median, mean and maximum concentrations of estrone in wastewater effluent were 13, 7.6, and 38 ng/L, respectively, for 12 municipal water treatment plants along the Thames River in Canada. In a survey of 18 municipal wastewater treatment plants across Canada, Servos et al. (2005) reported mean concentrations of 1.8 ng/L (range 0.2–14.7 ng/L) estradiol and 17 ng/L (range 1–96 ng/L) estrone in effluent.
96. Swartz et al. (2006) described the migration of organic ground water contaminants from septic systems. The authors found that estrogenic compounds decrease with distance from the septic system studied. The closer to a septic system, the higher the estrogenic compounds in ground water. Measured concentrations were 120 ng/L estrone and 45 ng/L 17 β-estradiol at 1.8 meters from the leach pits, and the concentrations decreased to 20 ng/L estrone and 3 ng/L 17 β-estradiol at a 6-meter distance from the leach pit. Haraughty (1999) reported that the 1990 census estimates suggest that over 27,000 septic systems are

[5] Secondary standards are non-enforceable guidelines regulating contaminants that may cause cosmetic effects (such as skin or tooth discoloration) or aesthetic effects (such as undesirable taste, odor, or color) in drinking water. EPA recommends secondary standards to water systems but does not require systems to comply.

in place in the 3 main Oklahoma counties of the watershed and estimated that 75% of these systems are substandard. Inadequacies may range from insufficient lateral lines, lack of or insufficient septic tanks, direct disposal of grey water to streams, ditches or land surfaces, and improperly located tanks and lateral lines. The estimated population of the Oklahoma counties in the IRW has increased dramatically since 1990 (U.S. Census Bureau Undated). See Figure 16. If the population is limited to only those living within the IRW, the same trend is observed (Figure 17) (Smith 2008). Hormones found in ground or surface water in the IRW are produced by multiple sources, and this is compounded by the increasing human population.

97. In contrast to the ng/L concentrations in drinking water reported by Dr. Olsen, women on hormone replacement therapy take milligrams, rather than nanograms (1 ng = 10^{-6} mg), of conjugated estrogens every day. Merck (2008) reports the most commonly prescribed oral doses as 0.3 or 0.625 mg conjugated estrogens, 0.625 or 1.25 mg estropipate, and 0.5 or 1 mg micronized estradiol-17β. Assuming one drinks two liters of water per day, the amount of hormones ingested, at the water concentrations reported by Dr. Olsen, would be about a million-fold less than the amount consumed every day by a woman on replacement therapy.
98. In summary, Dr. Olsen makes no claims of health effects from hormones found in Lake Tenkiller or springs or lakes of the IRW. The concentrations which Dr. Olsen reports are less than 2 ng/L. All animals, including humans, excrete hormones. There is no evidence to suggest that the hormone concentrations reported by Dr. Olsen are poultry-related.

CONCLUSIONS

99. Bacterial impairment of recreational waters occurs all over the State of Oklahoma; there is nothing to indicate such impairments are unique to the IRW. ODEQ in its 303(d) report claims multiple potential sources for the bacterial impairment of water bodies in the IRW and does not specify poultry as a source.
100. There is no evidence that bacteria from poultry litter have been associated with waterborne illness in the IRW.
101. The AWQC for bacteria are based on epidemiologic studies where the source of bacteria was sewage and not applicable to bacteria from poultry litter.

102. There is no evidence that the presence or concentrations of DBPs, cyanobacteria, nitrates, hormones, or metals in the waters of the IRW is unique in comparison to other parts of Oklahoma or the United States.

103. There is no evidence of a link between DBPs, cyanobacteria, nitrates, hormones, or metals and illness in the IRW.

ADDENDUM

I hereby adopt as a part of this report my previous testimony and report in this case.

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Figure 1. Campylobacteriosis Rate by County in Oklahoma, 2002 (OSDH 2003)

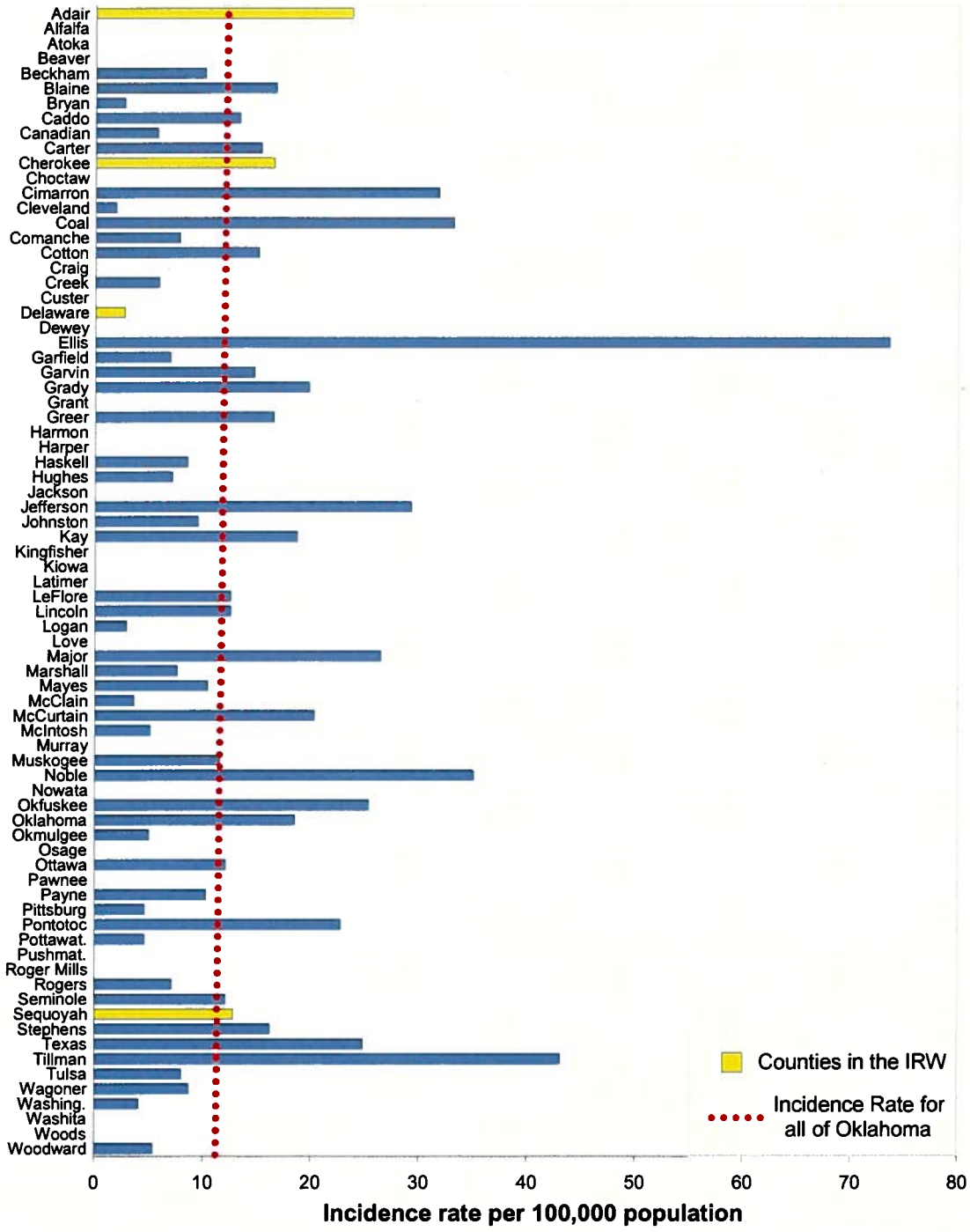


Figure 2. Campylobacteriosis Rate by County in Oklahoma, 2003 (OSDH 2004a)

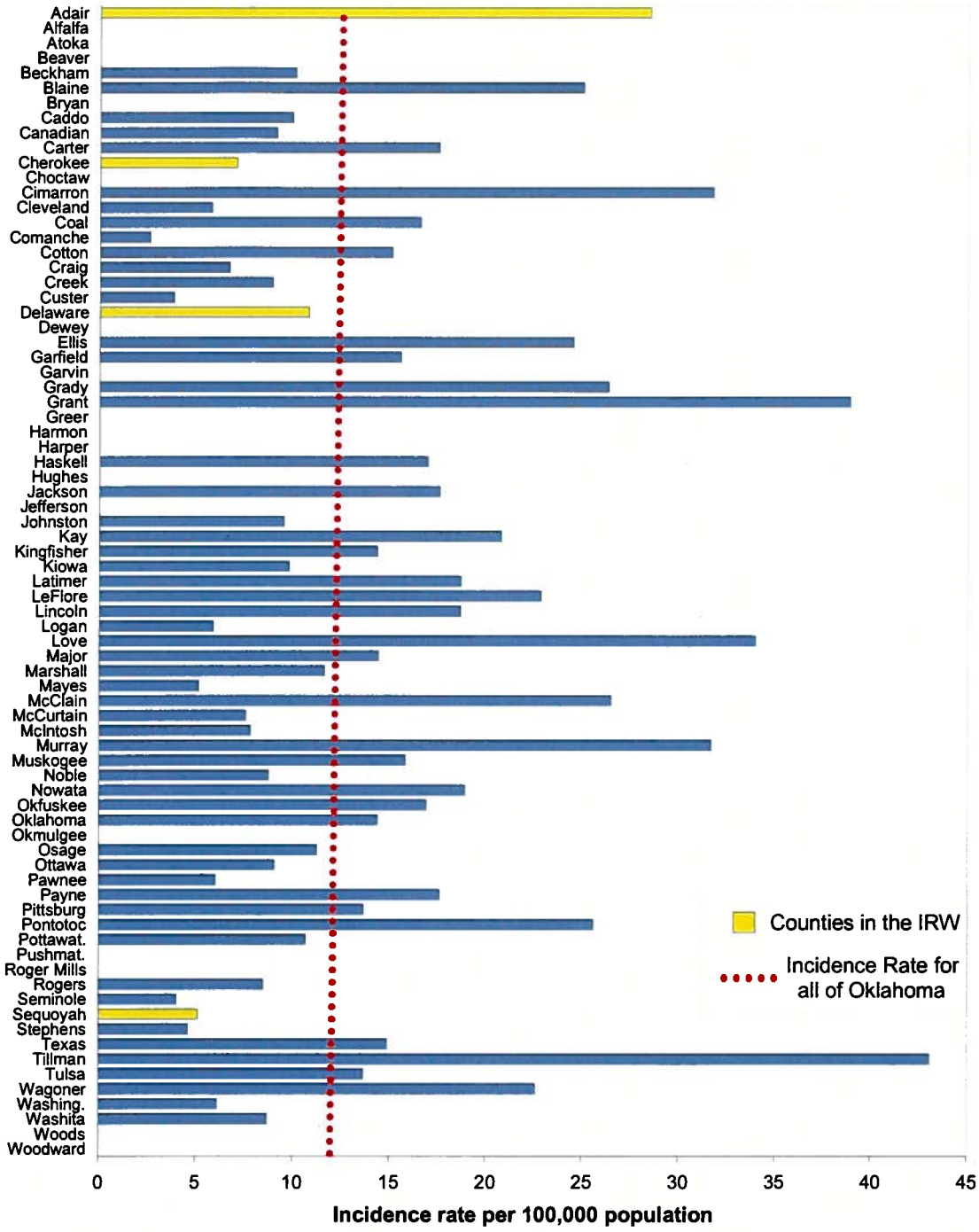


Figure 3. Campylobacteriosis Rate by County in Oklahoma, 2004 (OSDH 2005a)

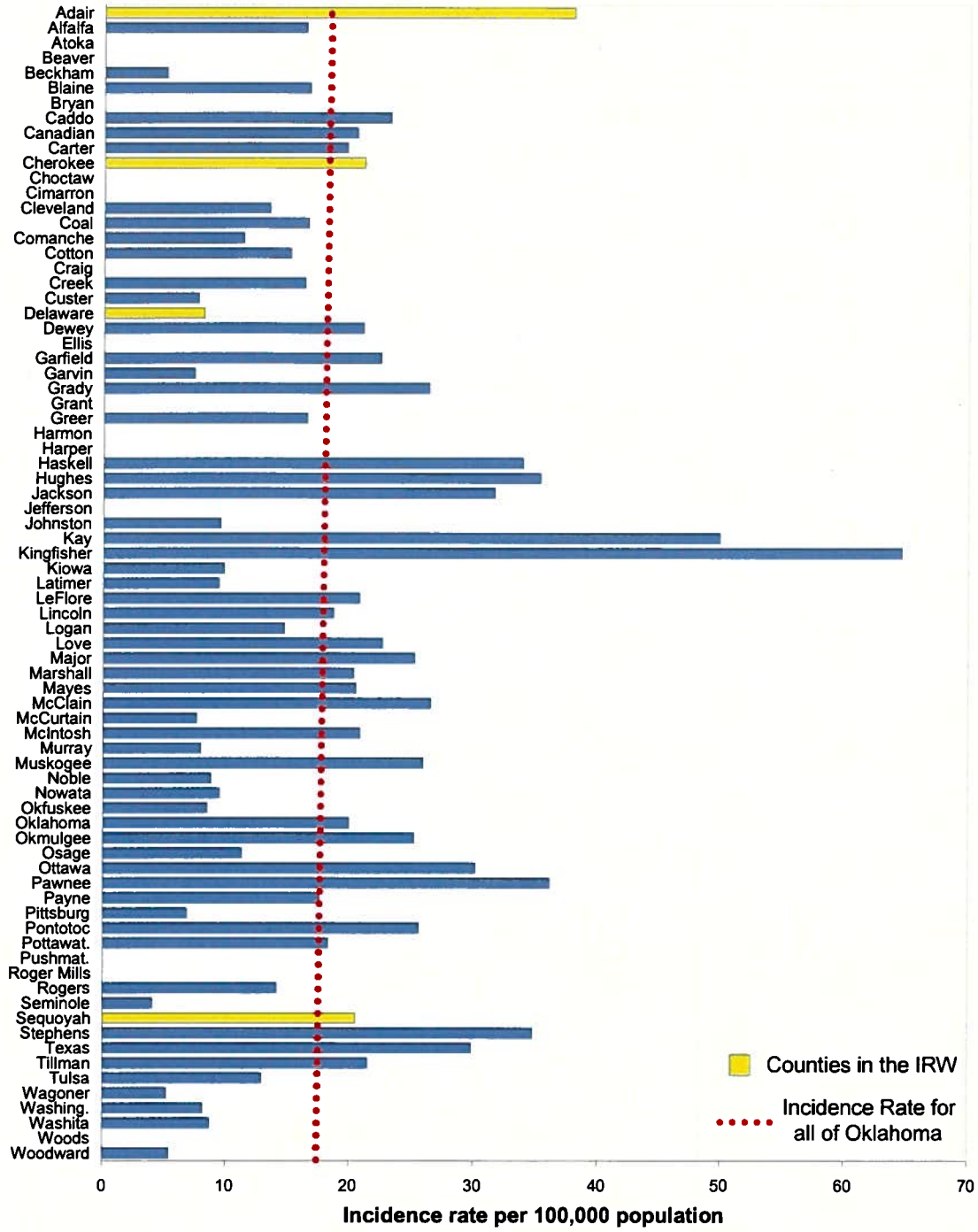


Figure 4. Campylobacteriosis Rate by County in Oklahoma, 2005 (OSDH 2006a)

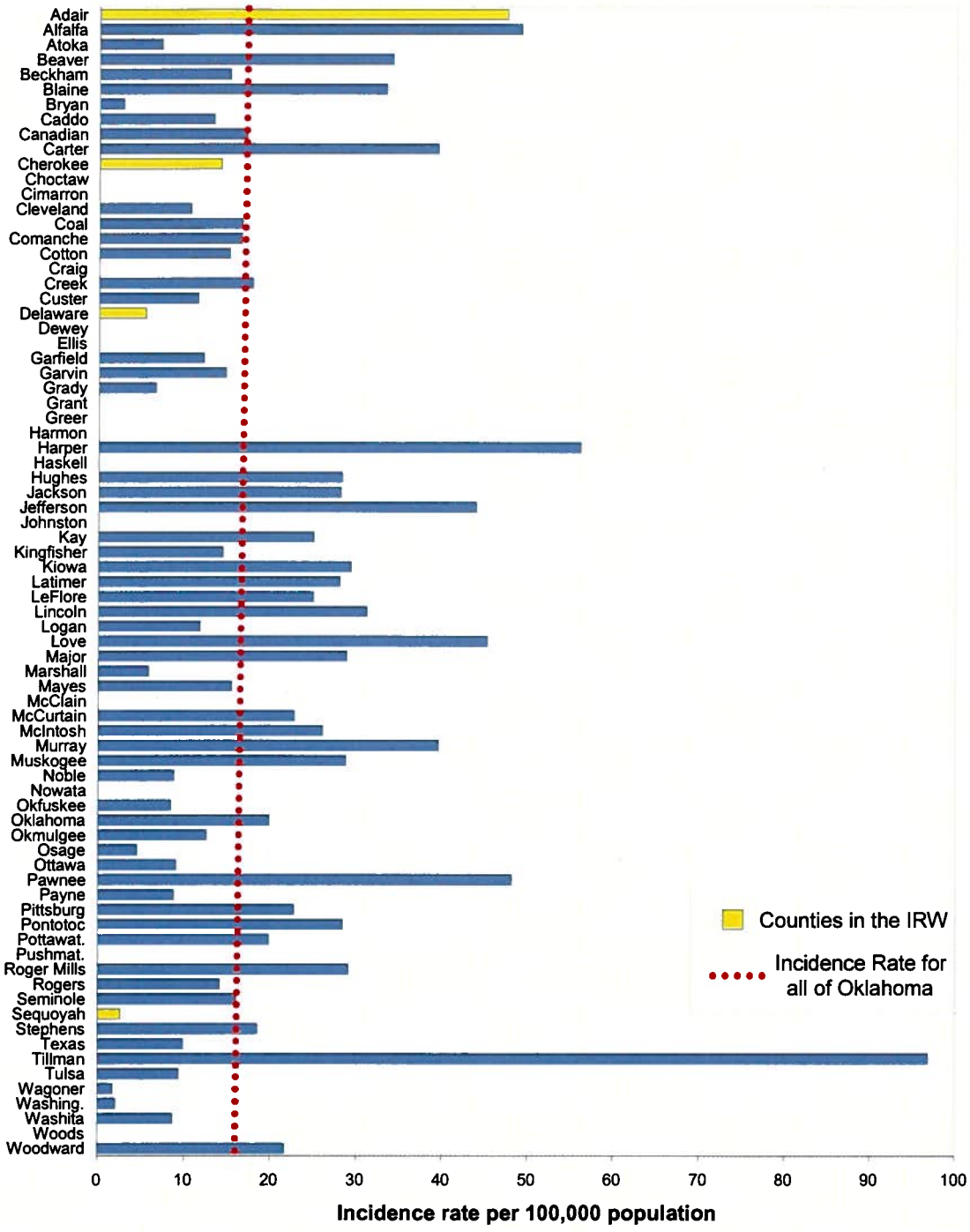


Figure 5. Campylobacteriosis Rate by County in Oklahoma, 2006 (OSDH 2007a)

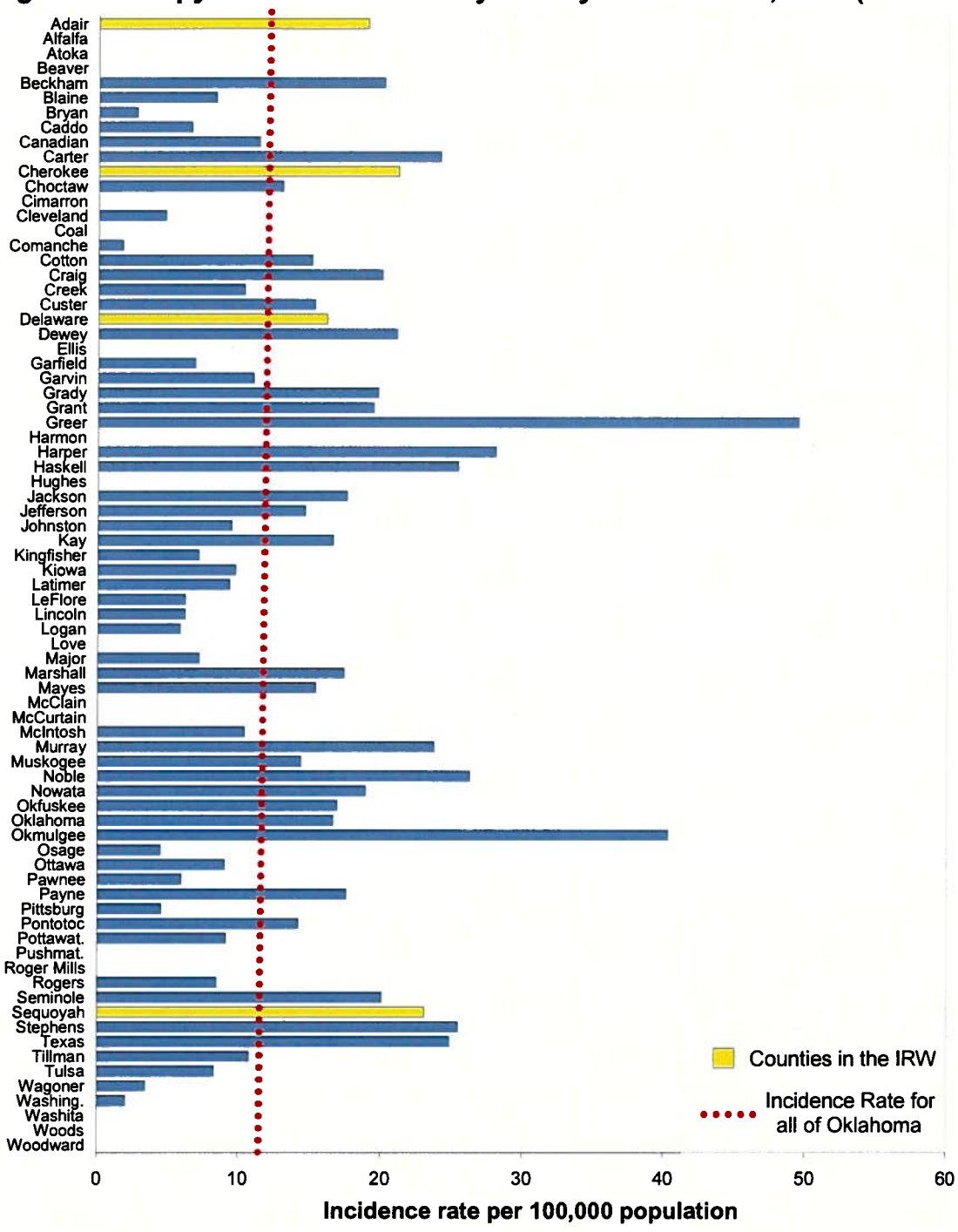


Figure 6. Campylobacteriosis Rate by County in Oklahoma, 2007 (OSDH 2008a)

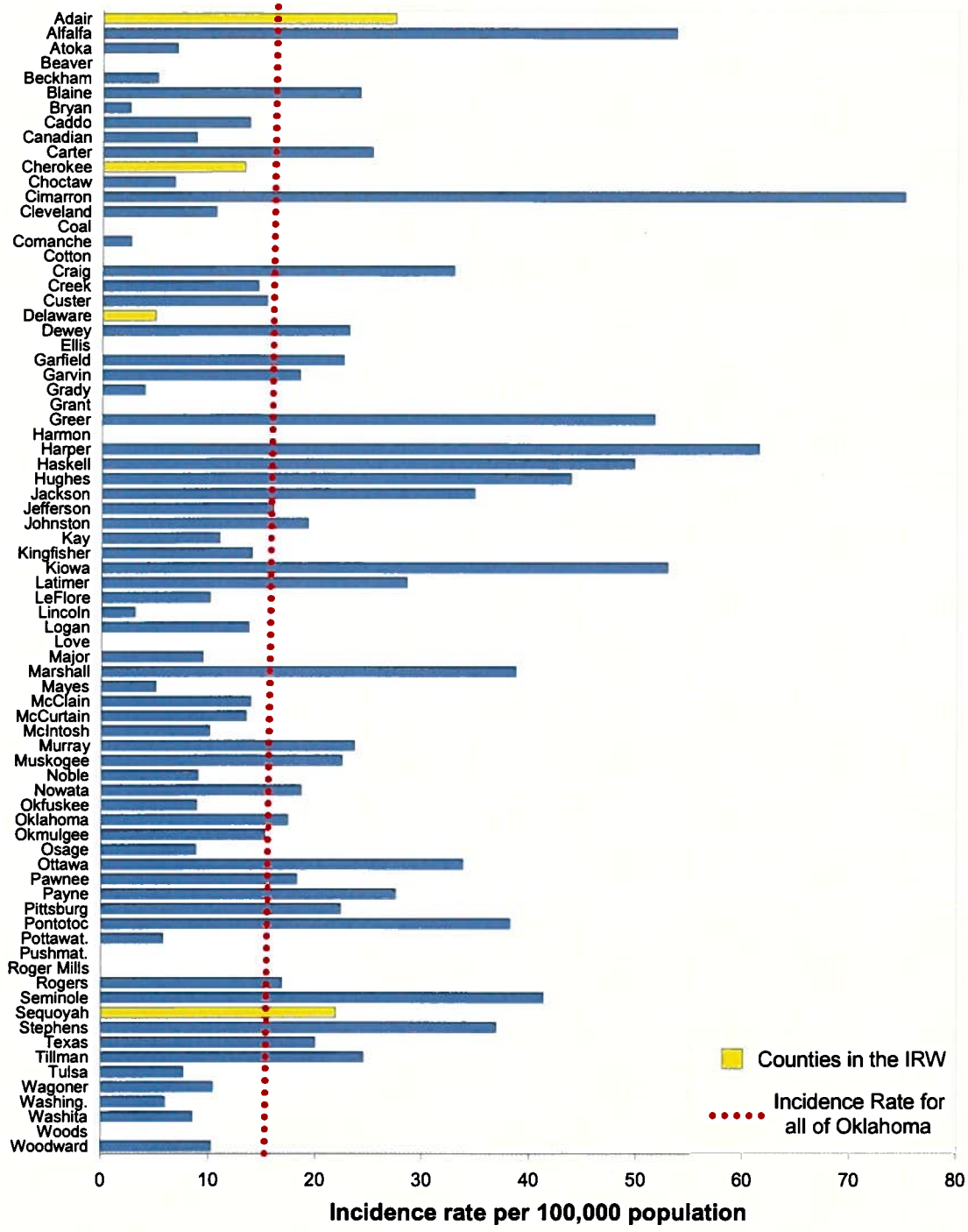


Figure 7. Salmonellosis Rate by County in Oklahoma, 2002 (OSDH 2003)

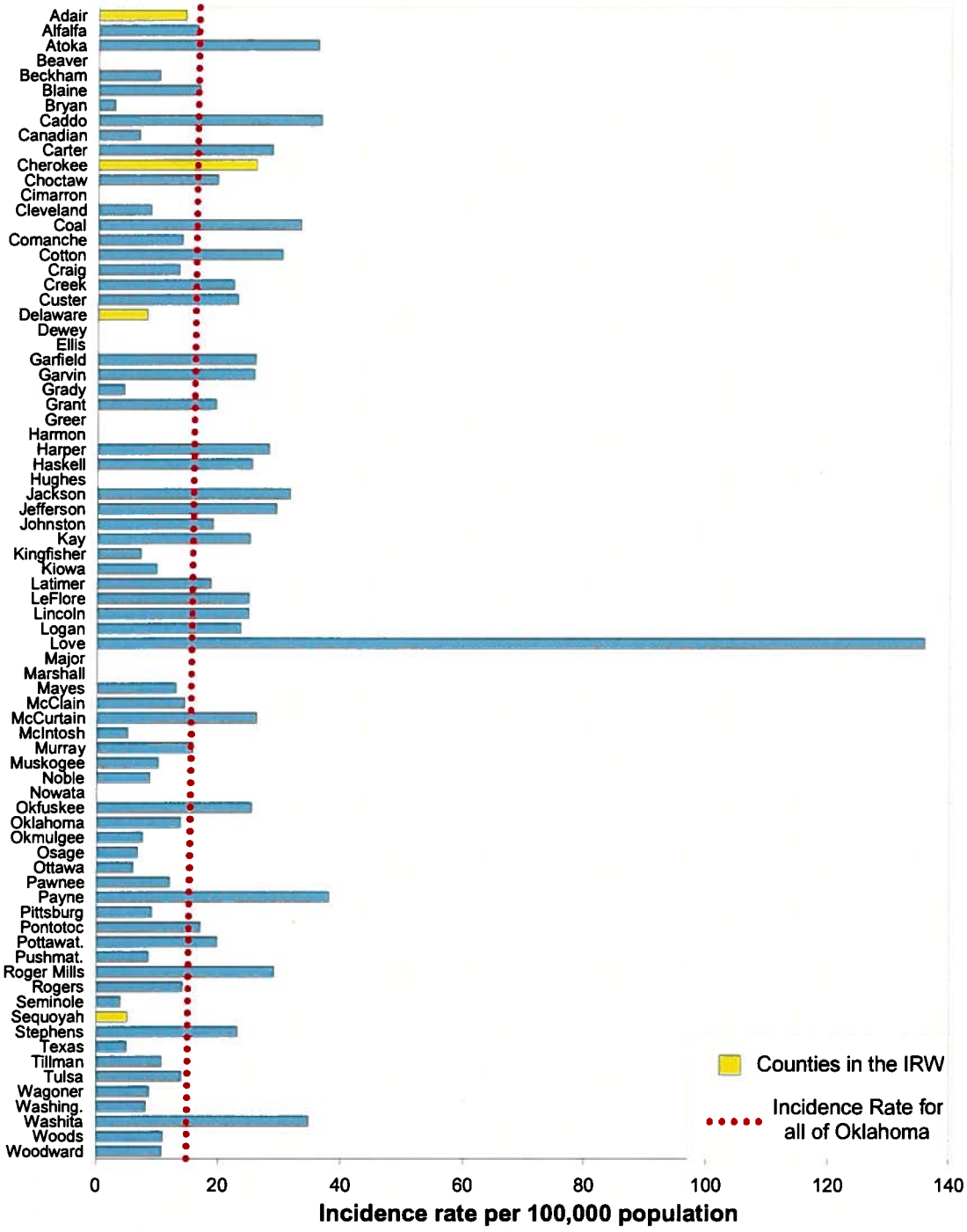


Figure 8. Salmonellosis Rate by County in Oklahoma, 2003 (OSDH 2004a)

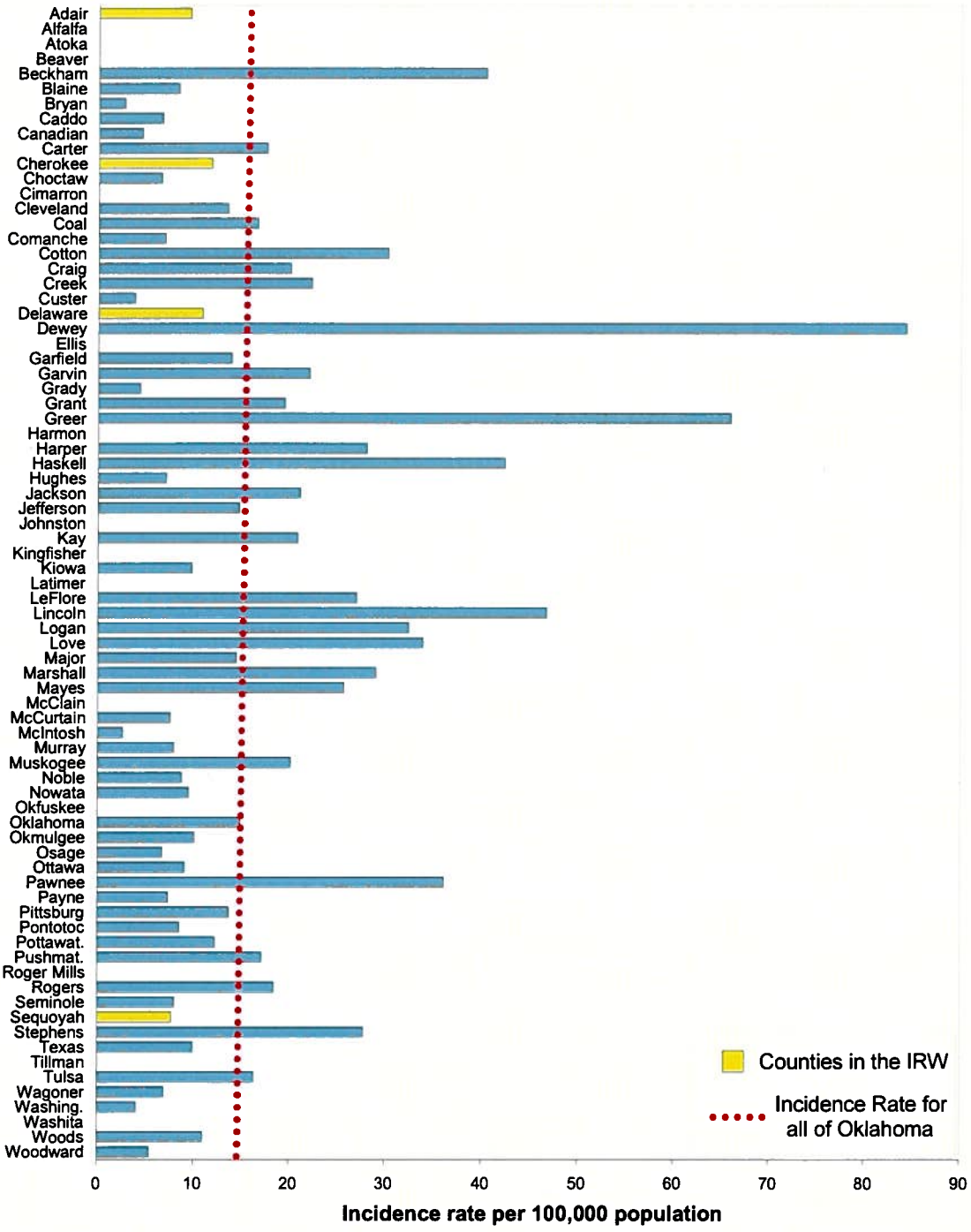


Figure 9. Salmonellosis Rate by County in Oklahoma, 2004 (OSDH 2005a)

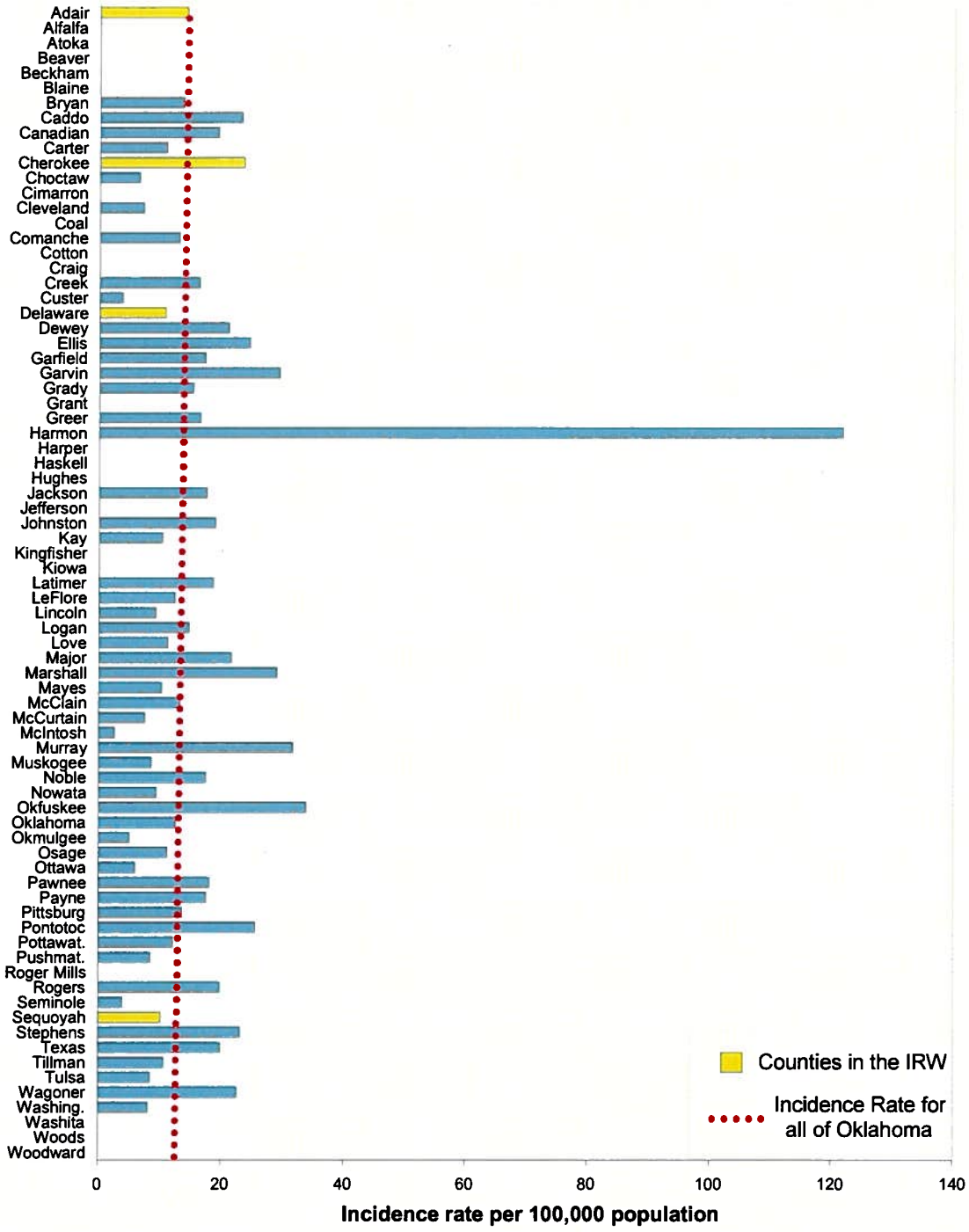


Figure 10. Salmonellosis Rate by County in Oklahoma, 2005 (OSDH 2006a)

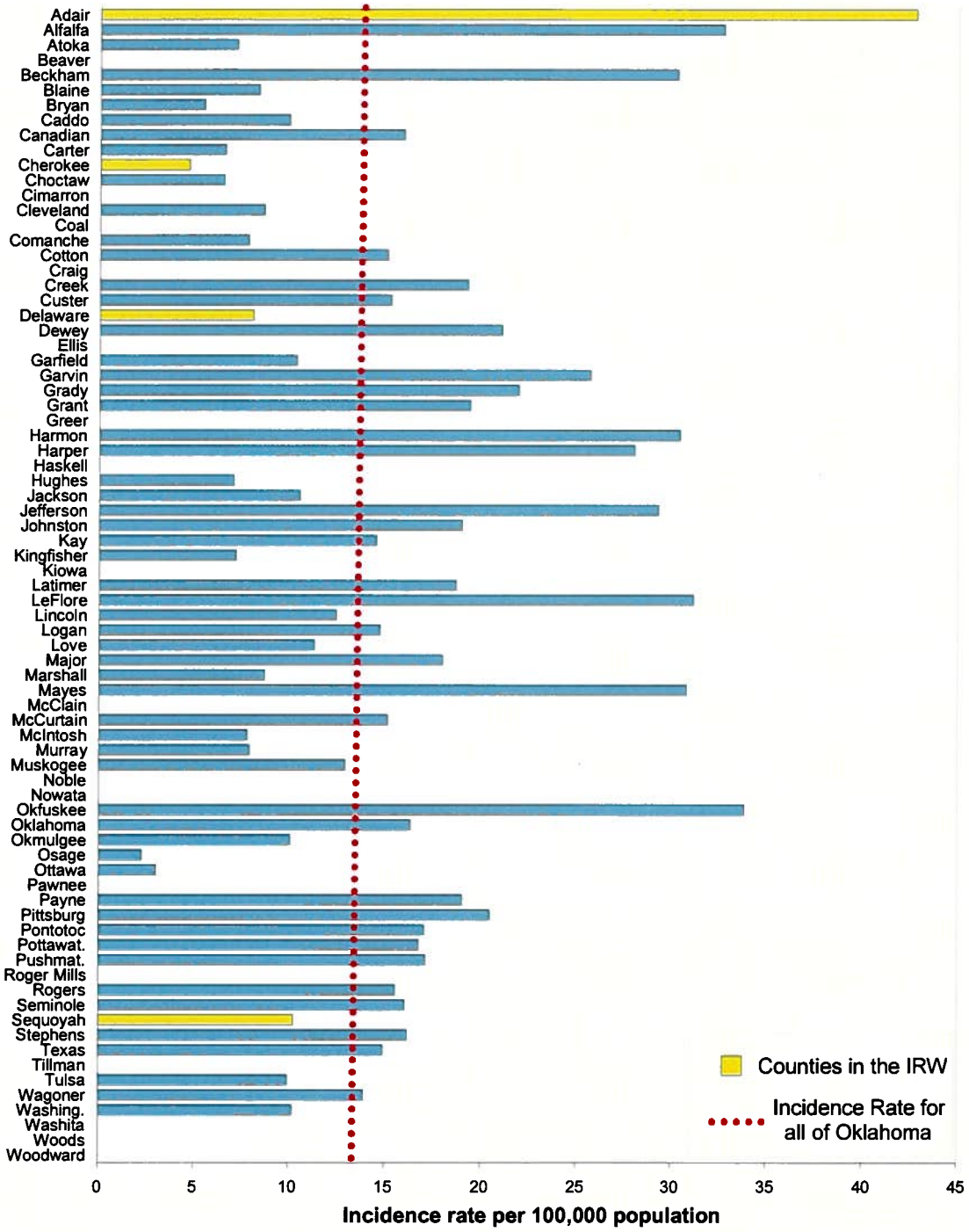


Figure 11. Salmonellosis Rate by County in Oklahoma, 2006 (OSDH 2007a)

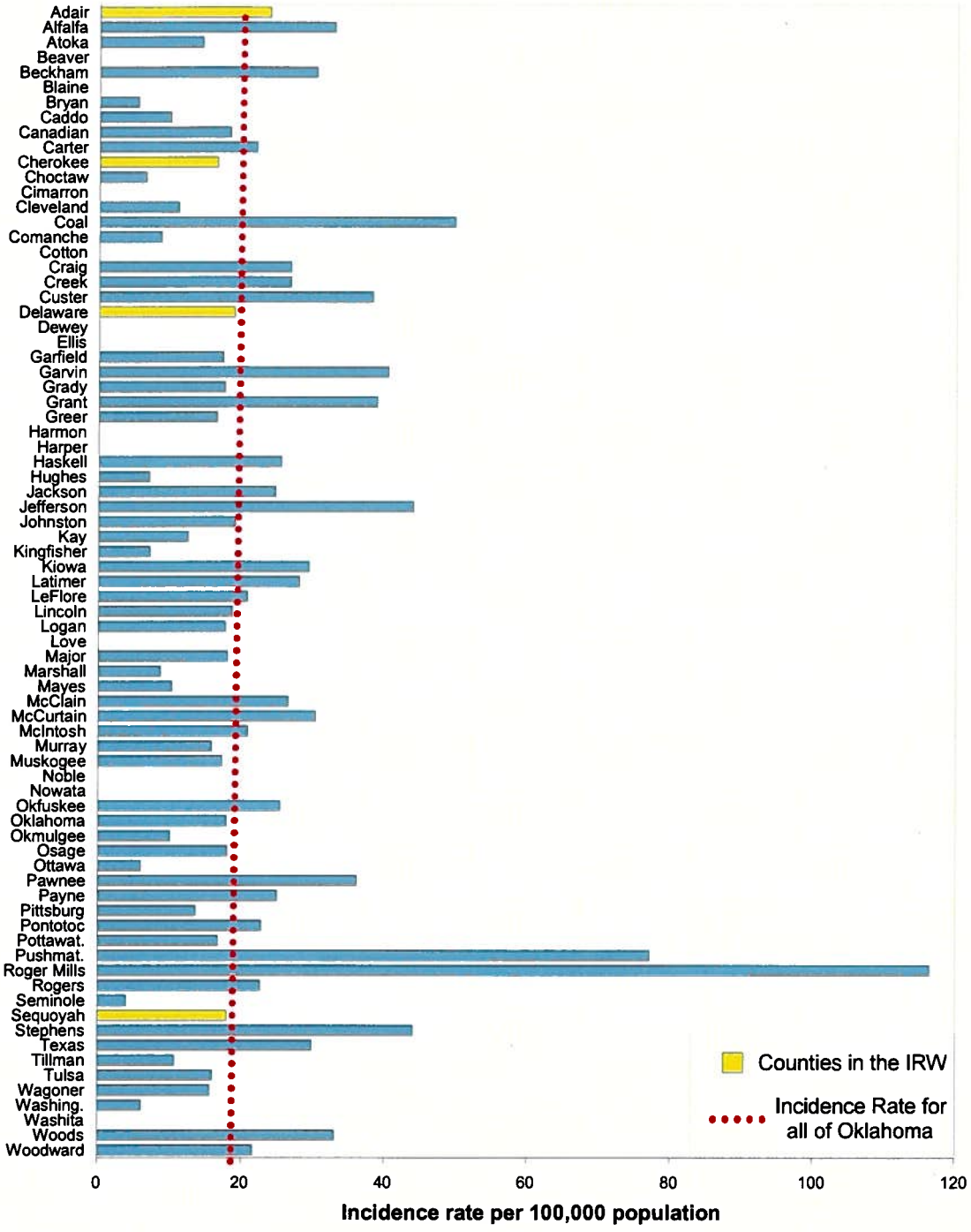


Figure 12. Salmonellosis Rate by County in Oklahoma, 2007 (OSDH 2008a)

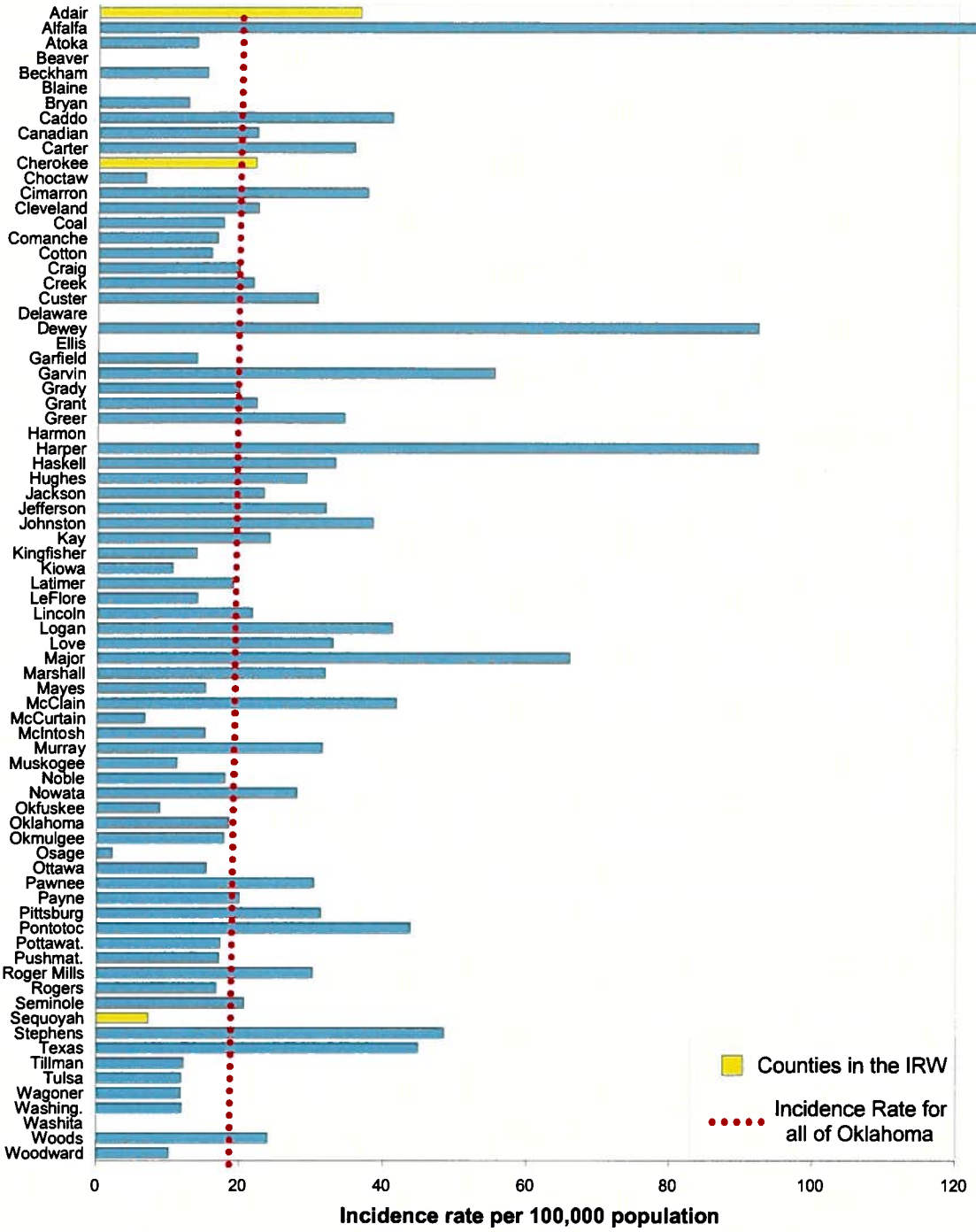
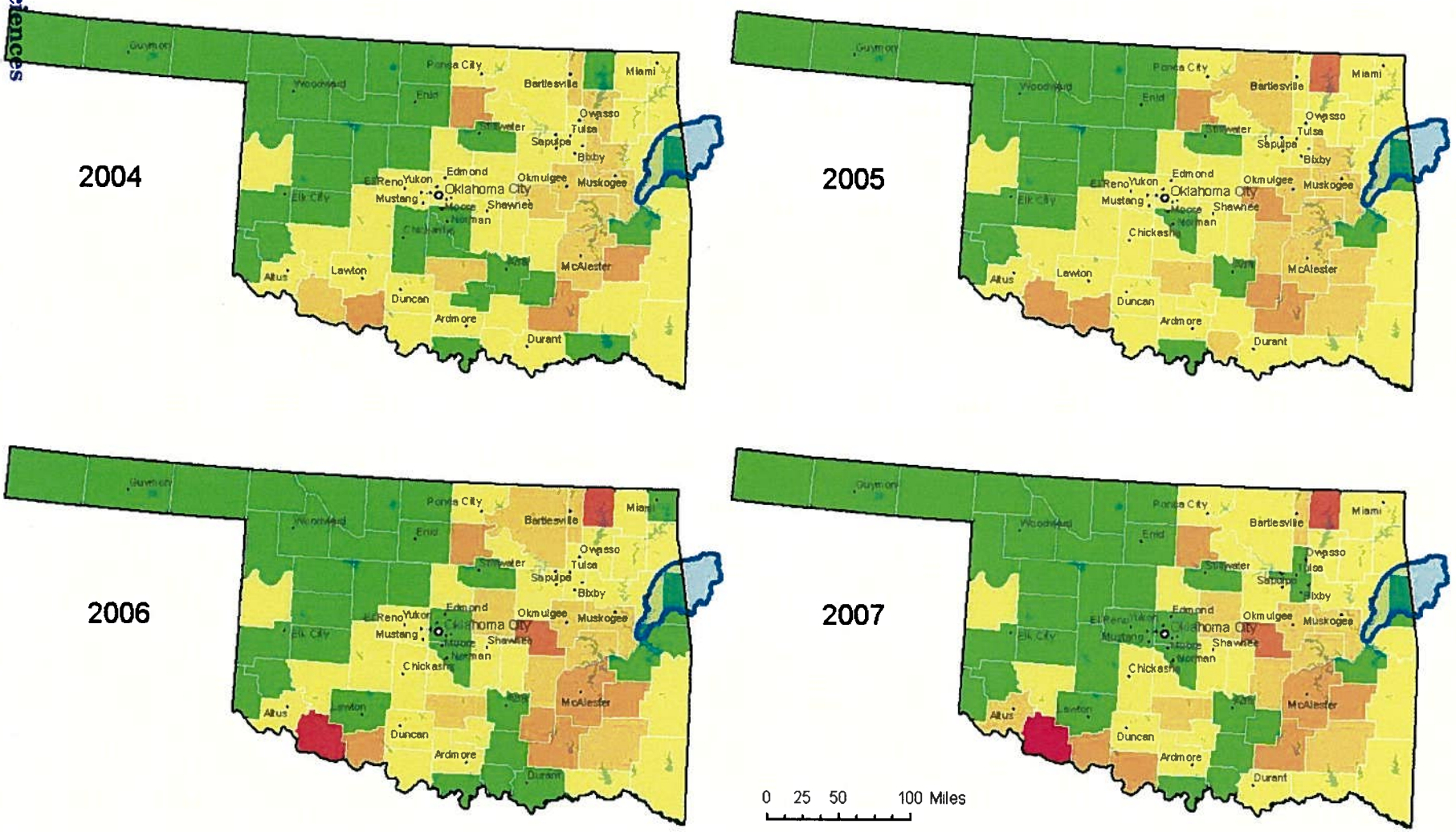


Figure 13. Percentages of Oklahoma Public Water Systems Having a DBP Violation of the MCL by Principal County Served, 2004-2007*



* Public water systems with DBP violations are listed in the Oklahoma Annual Compliace Reports (ODEQ 2008b, 2007, 2006b, 2005). The principal counties which these systems served was extracted from the ODEQ SDWIS database (ODEQ 2008c).

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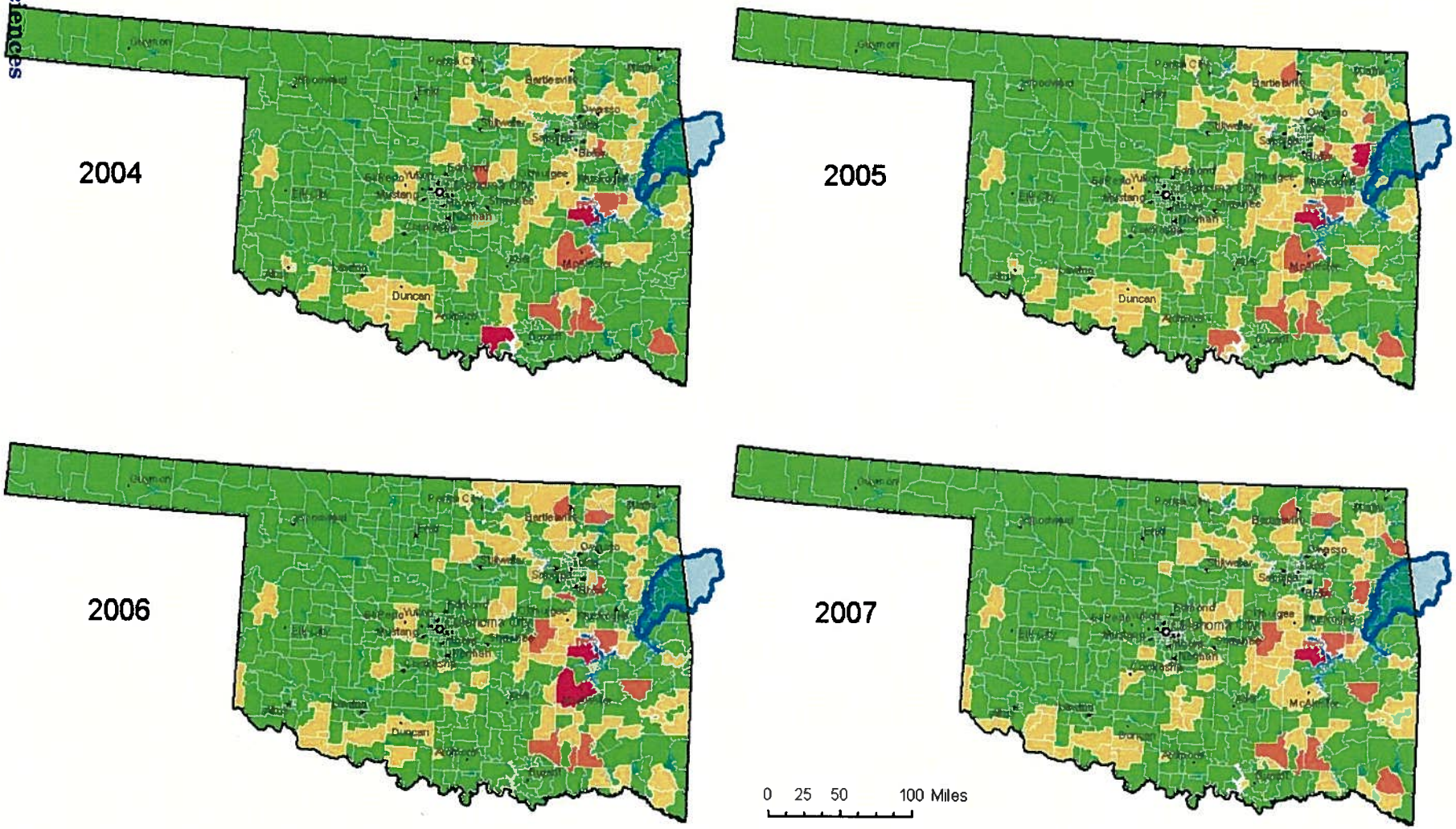
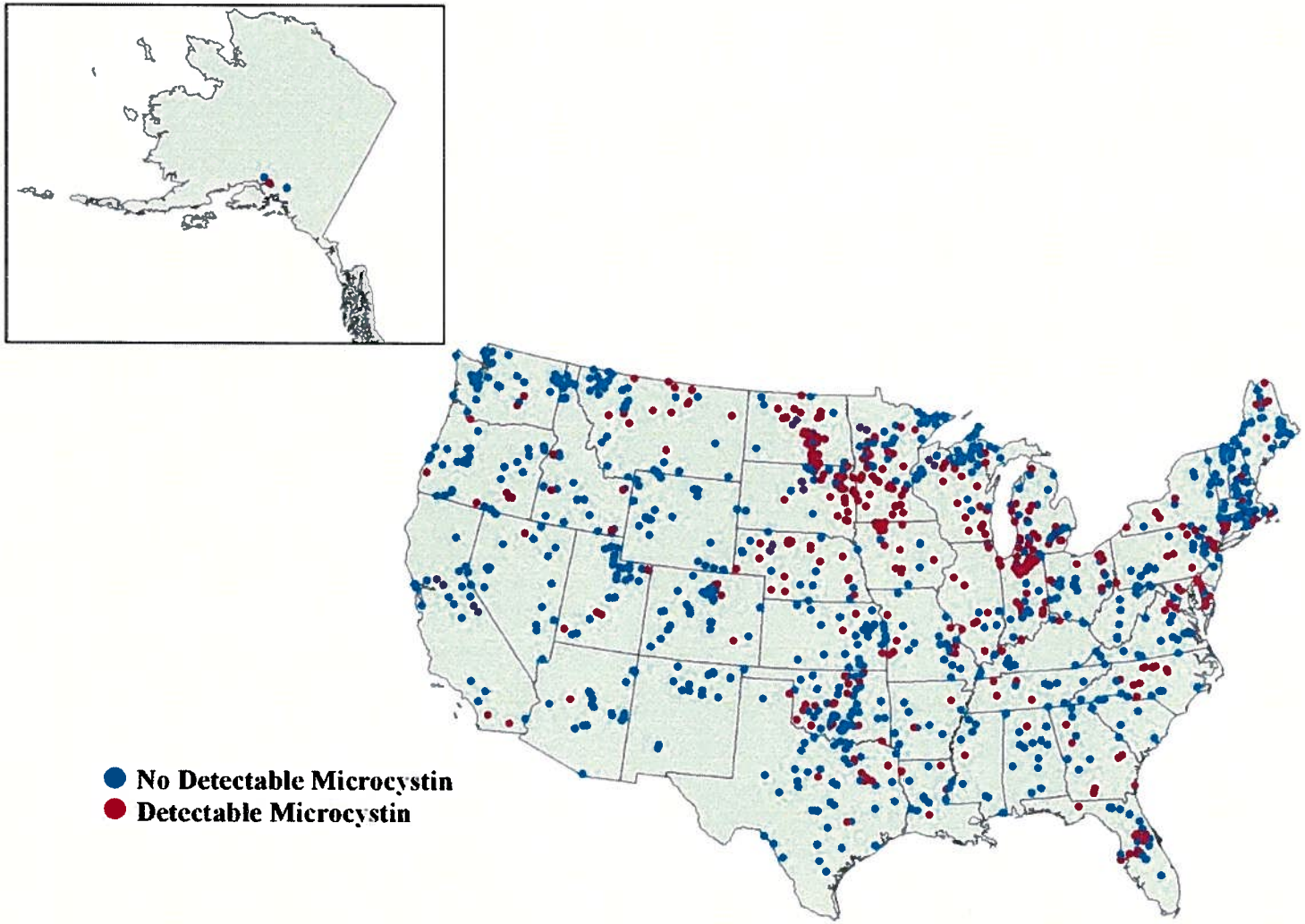
Figure 14. Number of Public Water Systems in Oklahoma Having a DBP Violation of the MCL by Zip Code, 2004-2007*



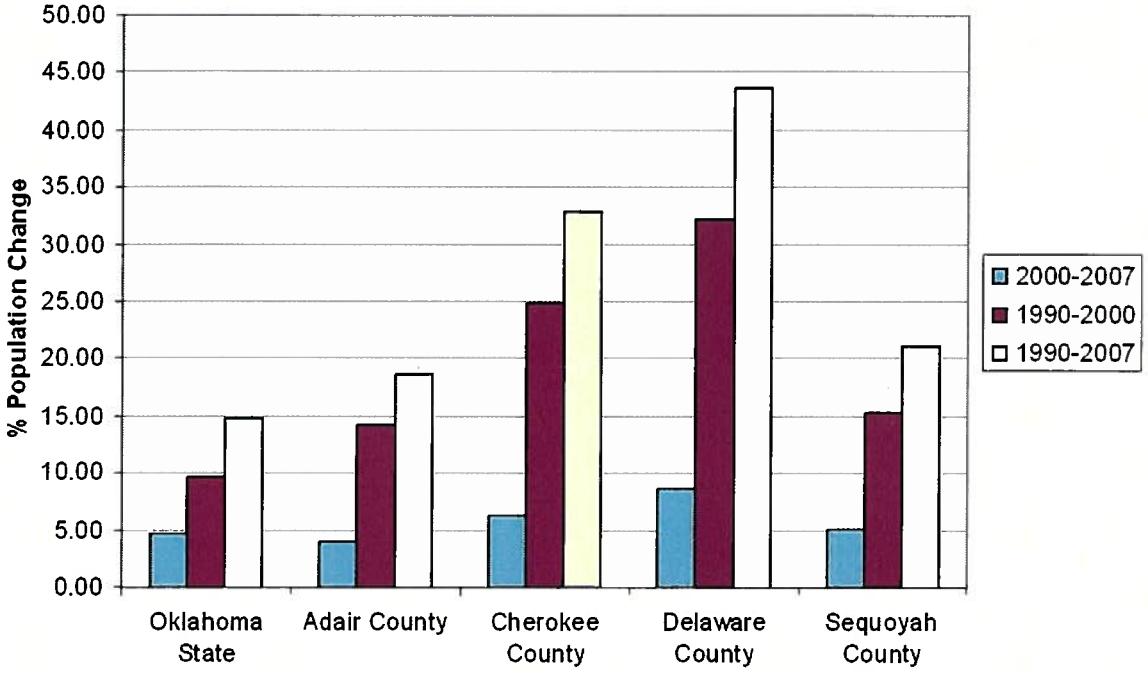
* Systems with Violations and their addresses, including zip code. are listed in the Oklahoma Annual Compliance Reports (ODEQ 2008b, 2007, 2006b, 2005).

Figure 15. 2007 EPA National Lakes Assessment, Microcystin Detections*



* From Loftin et al. 2008, slide 10. This study is currently under EPA review before publication.

Figure 16. Percent Annual Population Change for IRW Counties and Oklahoma State, US Census Bureau 1990-2007 Estimates*



*Estimates have been calculated using U.S. Census data from the 1990 and 2000 census (U.S. Census Bureau 2000, 2007)

Figure 17. Population Estimates within the Illinois River Watershed, 1950-2000 (Smith 2008)